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BLASTOMYCOSIS OF THE LARYNX*

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PHILADELPHIA

For more than a decade medical literature has been replete with descriptions of lesions in man due to certain yeastlike organisms commonly referred to as Monilia. A diligent search of the literature, however, has revealed only six cases in which the larynx apparently was the primary site of involvement. A case reported by Downing,[1] also by Dennis,[2] ultimately developed pulmonary involvement which proved fatal. The other cases were reported by Sartory, Petgès and Claouè,[3] Jackson,[4] New,[5] who reported two cases, and Casteran.[6] The paucity of reported cases would indicate that this disease is rare. Our experience, however, suggests that these cases are probably relatively common but are not always recognized as a fungus infection. This could be attributed to the erroneous belief that fungi found in bacteriologic cultures of secretions from the larynx are contaminants or that the tissue removed for histologic examination exhibits either a chronic laryngitis or a tuberculous lesion. This latter interpretation may account for the occasionally reported case of primary tuberculosis of the larynx.

Yeastlike fungi, like those isolated from our cases, are exceedingly common in nature. Ordinarily they are saprophytes, but some-

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times they assume parasitic habits and produce disease in individuals who are not robust. A classical example of this is the bronchomycosis of the Orient known as "tea factory cough," an infection acquired through the inhalation of fungi on tea leaves, by tea graders. Theobald Smith,[7] commenting on such accidental parasitism, remarked that "latent capacity for adaptation and opportunity to try them are responsible for many instances of strayed or aberrant parasitism . . . ." The disproportion between the host's resistance and the organism's capacity to adapt itself to its new environment seemed evident in our patients from the chronicity of the disease, the more or less granulomatous character of the lesions and their refractory response to treatment.

To Castellani[8] belongs the credit of stimulating interest in the blastomycoses of the respiratory tract and elsewhere. In his effort to differentiate between various "yeastlike fungi," he classified the subfamily Blastosporineæ, into genera, according to their capacity or lack of it, to produce ascospores and mycelium on suitable medium (vide infra).

BLASTOSPORINEAE

Ascospores present		Ascospores absent	
Mycelium present	Mycelium absent	Mycelium present	Mycelium absent
Endomyces	Saccharomyces	Monilia	Cryptococcus

His genus Monilia, to which the fungi reported here belong, was divided into species, chiefly by their ability to ferment various carbohydrates. Langeron and Talice,[10] Ashford[11] and Shrewsbury[12] restudied these organisms. They concluded that Castellani's "Monilia" are not monilia, since they do not conform with the genus defined by Persoon in 1801.

Langeron and Talice[10] suggested a new classification for them, based largely on the morphology of the organisms. Dodge,[13] approaching the problem from the standpoint of the botanist, includes them in the Eremascaceæ imperfecti and has again rearranged the genera. In spite of the taxonomic difficulties and the differences of opinion among mycologists, progress in the clinical recognition of specific blastomycoses, their prognosis and treatment will progress but little, unless careful detailed descriptions are given of the organism in each instance. We have tried here to do this rather than to give them a name.

The presence of a laryngeal lesion, exhibiting extensive chronic inflammatory changes more or less granulomatous in character in a

patient who presents no evidence of tuberculosis or syphilis should suggest fungus infection. In the three cases to be reported a tentative diagnosis of tuberculosis had been made before admission to the Bronchoscopic Clinic; this diagnosis, however, could not be substantiated after studies had been completed.

Case 1.—A man, aged 23 years, a sailor, was first seen in August, 1930, for relief of stenosis of the larynx. Ten months before admission to the Bronchoscopic Clinic he complained of hoarseness, which became progressively worse. Dyspnea developed and tracheotomy became necessary seven months following the onset of symptoms. During this time he was considered as a case of tuberculosis, although there was no evidence of a pulmonary lesion and repeated examinations of sputum were negative for tubercle bacilli.

When examined the laryngeal airway was almost completely obliterated by an extensive inflammatory infiltrative process which involved both vocal cords and ventricular bands. The vocal cords presented a granular surface; the edges were irregular, suggesting superficial erosions, and both were covered with a thin grayish-white exudate which extended to the subglottic area. There was swelling of the arytenoids and aryepiglottic folds with impairment of motility of both arytenoids. A tracheotomy cannula was in situ and the fistula was in good condition. The laryngeal appearances, although not typical, were suggestive of a tuberculous lesion. Studies for syphilis and tuberculosis were again repeated with negative results.

By direct laryngoscopy the findings by mirror examination were confirmed. Secretions and tissue were secured for study. The secretions were negative for tubercle bacilli on direct smear and by cultural studies. A diagnosis of chronic ulcerative laryngitis was made on the basis of the histologic findings and clinical appearances.

The patient was continued on a regimen of vocal and physical rest with intralaryngeal instillations of mentholated oil. The inflammatory and ulcerative changes in the larynx improved slowly, being replaced by stenosis. Direct laryngoscopic dilatation was performed and the patient was ultimately decannulated, seven months later. He continued well, although slightly dyspneic, for eight months, when he contracted an acute upper respiratory infection. Dyspnea became marked and tracheotomy again became necessary. The larynx now presented an inflammatory appearance with definite ulceration of both vocal cords, particularly anteriorly. Grayish white exudate was present. There were noted infiltrative changes involving the tracheal wall. Thick tracheal secretions proved troublesome. A direct

laryngoscopic examination was carried out and secretions and tissue were secured for study.

A fungus found in the secretions presented the following characteristics:

Blastospores polymorphous, spherical, ovoid or elongate. Older cells stain more or less uniformly with iodine. Hyphæ septate, little branching. Terminal hypha consists of an elongate element. Gram positive, not acid fast. Fat demonstrable in yeast cells. No ascospores observed (Fig. 1).

Cultivation.—On Sabouraud's glucose agar, colonies are convex, smooth, creamy white, glistening, margins regular, averaging about 3 mm. in diameter in 96 hours. Glucose broth-pellicle not formed, granular, white sediment, liquid clear.

Biochemical Reactions.—See table for fermentations. Indol not formed. Milk unchanged. Serum not digested. Gelatin, inverted pine tree growth; liquifaction begins after 90 days. Optimum temperature, 37 degrees C.

Pathogenesis.—Killed young rabbits in three weeks, producing gray, dull, irregular nodules, 2 mm. in diameter, in liver and lungs. Fungus was demonstrable in lesions; both yeast and hyphæ were present (Fig. 2). In the patient's larynx it produced a chronic inflammatory lesion characterized by edema, ulceration, fibrosis and infiltration with polymorphonuclear leucocytes and round cells. Granulation tissue was abundant.

Identity.—It was classified at the time of study as one of Castellani's "Monilia."

The patient was given potassium iodide in increasing doses until 3 drams daily were taken. Some improvement in the laryngeal appearances was noted, but the cultures never were negative for fungi. Later sodium iodide, grains 30, was given intravenously with no additional improvement. Tartar emetic, in 1 per cent solution, was administered intravenously in ascending doses with no apparent benefit. The patient was then given ethyl iodide inhalations, beginning with one-half cc. daily and increasing the dose until 2 cc. daily were taken. There was noted definite improvement with this form of medication, but the patient developed symptoms of ataxic paraplegia necessitating discontinuance of the ethyl iodide. He has recovered from the paraplegia. Cultures from laryngeal and tracheal secretions still contained moniliform fungi. On the basis of observations by New[5] and Gill,[14] who reported beneficial results from roentgen treatment, this form of therapy was instituted. Improvement has been slight; the laryngeal lumen is practically obliterated by cicatricial tissue, although this cannot be attributed to the roentgen treatment. Curiously, repeated cultures from secretions and tissue have been negative for the organism. The patient is able to carry on at the present time as a caretaker. He is troubled with thick mucoid secretion and crusting. The lesion remains limited to the larynx and upper trachea. The lungs are negative. There is no skin involvement.

Case 2.—Man, aged 54 years, a tanner, developed hoarseness during February, 1934. This became progressively worse. A roentgen study of the chest showed an old tuberculous lesion apparently healed. He was placed on a regimen of vocal rest, with little change in the hoarseness. He was referred to the Bronchoscopic Clinic, fifteen months after the onset of symptoms. When admitted he was very hoarse and moderately dyspneic. There was some weight loss. By mirror laryngoscopy there were observed extensive inflammatory changes, swelling of the vocal cords and interarytenoid infiltration. All of these areas presented an irregularly nodular surface covered with grayish exudate with an occasional whitish elevation (Fig. 3). These suggested sago-like bodies on a grayish background. A fungus infection was suspected. Completed studies were negative for tuberculosis and syphilis. At direct laryngoscopy the findings by mirror examination were confirmed. The lesion was found extending subglottically beyond the edge of the vocal cords for a distance of several millimeters where it was sharply demarcated by an inflammatory zone. Tissue and secretions were secured for study. The histologic diagnosis of the tissue removed was chronic inflammatory laryngitis with ulceration. An anascosporous yeast was discovered. It presented the following characteristics:

Blastospores in most media are ellipsoid and doubly contoured, averaging about five microns in their longest diameter. In beerwort agar and on potato water they are distinctly elongate with pointed tips, lacrimiform or stalagmoid in character, measuring about six microns in length. They reproduce by budding dichotomously, producing small bushy masses. The hyphæ are septate, branched and are tipped by an elongate element or a blastospore, or several in a chain. At the margin of the beerwort colony, they are arranged in dense parallel masses. Cells are Gram positive, not acid fast and contain one or more droplets of fat. Fat is also present in the young hyphal cells. No ascospores on carrot, Gorodkova's agar or block of plaster of paris after 60 days.

Cultivation.—On Sabouraud's glucose agar, growth is even, slightly dull, thin, yellowish white and moist. Giant colony is thin, flat, dull, yellowish white, with a central eminence and radial striations and measures five cms. after 30 days. Margin slightly wavy, but not lobate. Colonies on beerwort agar are flat, raised, dull, yellowish white with a denticulate margin after one week. Glucose broth, turbid, becoming clear, with slight sediment and no pellicle or ring. Potato water, clear, slight flocculent sediment. Beerwort, turbid, becoming clear, with heavy, flaky sediment. Bichemical reaction, optimum temperature 37 degrees C. Indol not formed. Milk acid, no coagulation. Serum not digested. Gelatin growth consists of numerous fine, white points which become fuzzy; no liquifaction at the end of 60 days. On carbohydrate media liquid clear, no pellicle. See table for fermentation.

Pathogenesis.—Young rabbits inoculated with 1 cc. of suspension are killed in fifteen days. Viscera are congested. There are numerous gray-white nodules in the liver and lungs. Fungus is present in lungs and liver as a highly branched

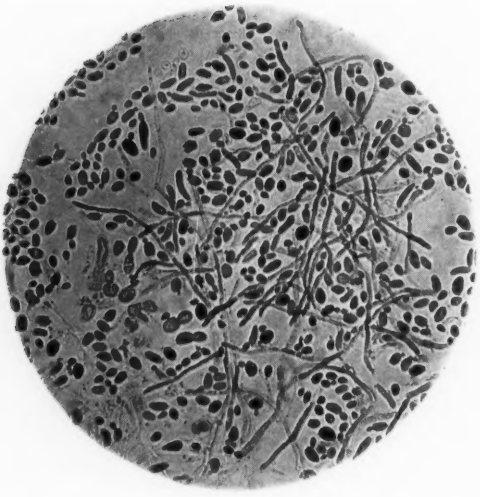

Fig. 1. Photomicrograph of fungus illustrating character of hyphae and blastospores (x500).

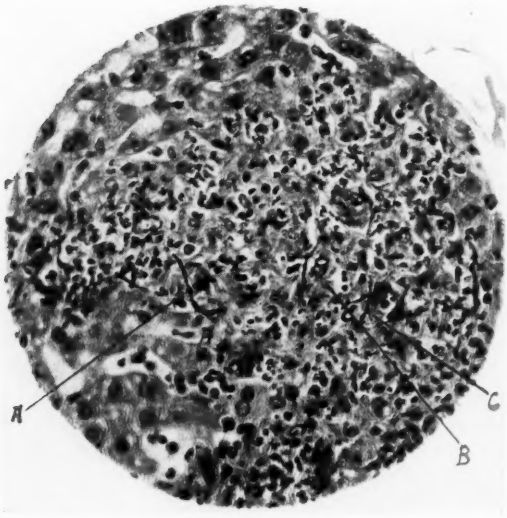

Fig. 2. Photomicrograph of liver of rabbit following inoculation with a pure culture of the organisms isolated from the patient's larynx. (x900)

septate structure. In the laryngeal lesion of the patient there is a chronic inflammatory change. The covering epithelium is slightly hyperplastic. The submucosa is edematous. Fibrosis is excessive and there is a moderate infiltration with cells of inflammatory origin.

Identity.—This organism is classified in the genus Blastodendrion, on the basis of the character of the giant colony, the type of blastospores and their arrangement.

Potassium iodide was administered in increasing doses with marked improvement in the appearances of the larynx, decrease in dyspnea and a gain in weight. This continued for eight months. At the end of that time the interarytenoid area appeared practically normal, and the vocal cords, particularly the right, exhibited improvement. The patient then contracted an acute infection which resulted in a prompt return of the inflammatory changes in the larynx together with an increase in the amount of exudate on the vocal cords. Iodides were continued in increasing doses with but slight improvement. There is a return of dyspnea with cough; the entire circumference of the glottic chink is surrounded by a nodular infiltrative process covered with grayish exudate. Secretions contain a yeastlike fungus.

Case 3.—Man, aged 53 years, a truck gardener, had been hoarse for a number of years, but this did not become marked until four years ago, when he noticed some shortness of breath on exertion. A diagnosis of pulmonary tuberculosis with laryngeal involvement was made and he entered a hospital for treatment. After eight months it was decided that the condition was not tuberculosis. The dyspnea became more marked and a tentative diagnosis of carcinoma was made, but this could not be corroborated by biopsy. Because of the dyspnea, tracheotomy became necessary. A diagnosis of chronic laryngitis was made. The patient was comfortable, but he could not dispense with the tracheotomy tube. Later, secretions and crusts became troublesome and excoriations of the skin about the tracheotomy fistula developed. He was referred to the Bronchoscopic Clinic for treatment. By mirror laryngoscopy, there was found an extensive infiltrative process involving the entire right side of the larynx, posterior extremity of the left vocal cord and the interarytenoid area. The anterior portion of the left cord could not be visualized because of an overhanging ventricular band. The arytenoids and the left ventricular band were swollen and edematous. The infiltrated area presented a granular appearance which suggested small nodules covered with grayish exudate. A tracheotomy tube was in situ. The fistula was surrounded by a purplish red zone which presented superficial erosions covered with exudate. The lungs were negative for

Fig. 3. Drawing shows a mirror view of the larynx in the case of a primary fungus infection of the larynx. The infiltrated vocal cords and interarytenoid tissues presented a nodular appearance and were covered with a thin grayish exudate and an occasional whitish elevation.

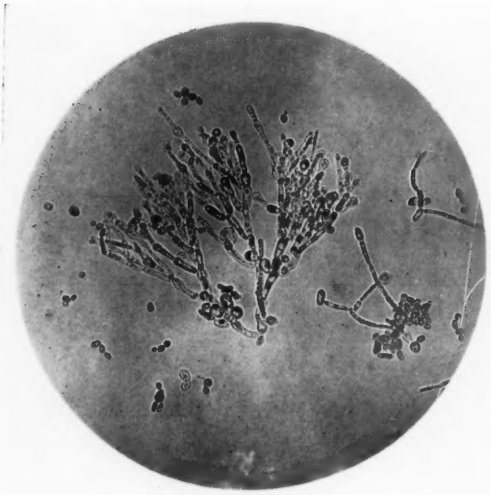

Fig. 4. Photomicrograph of fungus classified as belonging to genus Candida from culture on potato water (x500).

evidences of infection. The Wassermann was negative. Secretions and tissue from the larynx, also scrapings from the skin about the tracheotomy tube were secured for study. From all of these fungi were repeatedly recovered.

Description of the Fungus.—Blastospores are thick-walled polymorphous, spherical, oval, fusiform and asymmetrical, and measure four to six microns in diameter. The morphology is most varied on beerwort. There are definite blasto-arthospores present in this medium. These measure six microns, are elongate and often fusiform, some like a racquet. The blastospores sometimes occur in short chains or the spores are intercallated. It is occasionally difficult to differentiate between blastospores and portions of hyphæ (blasto-arthospores) (Fig. 4). Hyphæ septate, not easily disrupted, moderately branched, ends flattened, or like a drumstick, measure fourteen to fifty microns by two to three microns. Hyphæ terminate in elongated elements, a thick-walled blastospore, a short chain of blastospores or a verticil; verticils irregularly spaced when present (Fig. 5). Colony begins by budding of thick-walled blastospore. Coremia present. Gram positive, not acid fast. Yeast cells contain one or more excentrically placed globules of fat usually adjacent to a central vacuole. No ascospores on carrot, Gorodkova's agar or plaster of paris blocks after seventy days.

Cultivation.—Growth on Sabouraud's glucose agar, thick, yellowish white, dull, membranous, tenacious, becoming wrinkled and a deeper yellow. Growth on beerwort agar, yellow, membranous, tough, dull and covered with hyphæ, giving a felt-like appearance.

Giant colony.—Thick, yellowish white, membranous, tenacious, concentrically ringed with central eminence and lobate. Numerous warty excrescences appear about the center of the colony with some wrinkling after sixty days (Fig. 6). Glucose broth pellicle forms, which sinks to bottom and a new one forms; liquid clear, with white, ropy sediment. Potato water, surface contains few thin islets of fungus; liquid clear, flocculent sediment. Beerwort, thick, yellowish white pellicle, turbid, clears, forms thick tenacious sediment.

Biochemical reactions.—Optimum temperature, 37 degrees C. Gelatin, inverted pine tree growth, stratiform liquifaction after 60 days. Indol, not formed. Serum, not digested. Carbohydrate media, pellicle, ring and sediment present, liquid becomes clear. When fermentation occurs the gas is later absorbed and the medium becomes alkaline. See table for fermentations of carbohydrates.

Pathogenesis.—Young rabbits are killed in fifteen days when given 1 cc. of suspension of fungus intravenously. Viscera congested, gray with irregularly shaped, discrete, raised nodules in liver and lungs. When opened they exudate a thin, milky fluid. Both yeast forms and hyphæ are demonstrable in the lesions. In the tissues they are highly branched. The foci are necrotic and are infiltrated with cells of inflammatory origin.

The lesion in the patient's larynx is characterized by hyperplasia of the covering epithelium, areas of ulceration and chronic inflammation. In the submucosa there is considerable edema. There are many giant cells present, some of which contain the yeastlike fungi. The tissue is quite vascular and is richly infiltrated with cells of inflammatory origin and in some respects resembles an atypical tuberculous lesion (Fig. 7.).

Fig. 5. Photomicrograph of edge of colony on a slant of glucose agar illustrating branching and irregularly spaced verticils. The black masses consist of yeast-like cells (X80).

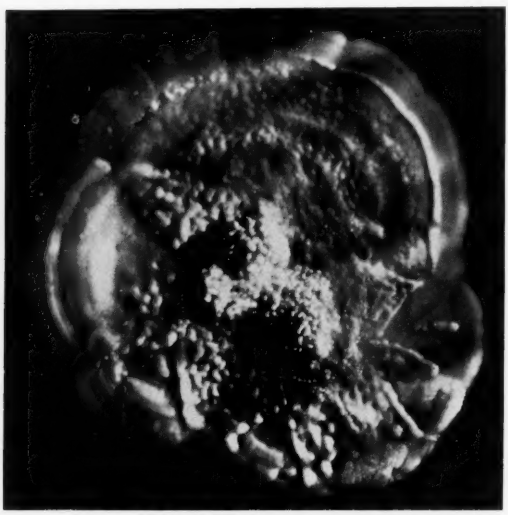

Fig. 6. Photograph of giant colony after 40 days of growth.

Identity.—The membranous colony, presence of blasto-arthrospores, fermentation of carbohydrates, pellicle formation and finer morphologic structure of this organism resemble those of the genus Candida. The lobed margin of the colony and liquifaction of gelatin do not conform to Dodge's[13] description. However, one of these organisms, C. Krusei, is distinctly lobed in Shrewsbury's[12] illustration of it, and liquifaction of gelatin, according to Langeron and Talice,[10] is an individual factor rather than an identifying characteristic. It is placed in this group for the present.

The bacteriologic findings of a pathogenic fungus, absence of tubercle bacilli, negative chest findings for tuberculosis, the appearances of the larynx and the skin about the tracheotomy fistula, with histologic findings of a chronic ulcerative lesion, favored the diagnosis of fungus infection. Potassium iodide therefore was administered in increasing doses with very definite improvement. The skin lesion cleared promptly. The larynx exhibited improvement and the voice was better.

The patient's present condition is quite satisfactory, although secretion and crusting are troublesome. Decannulation has been advised against for this reason, although a complete cork can be worn during the daytime. Recent bacteriologic studies of secretion from the trachea show the persistence of a fungus. Irradiation therapy has not been carried out, but will be recommended.

Two additional cases have been examined recently in which yeastlike fungi have been found. Organisms found in secretions removed from the larynx in the case of a man, aged 39 years, a waiter, who complained of hoarseness, sore throat and dyspnea for about eight weeks following an acute cold, were described as follows:

Morphology.—Yeast cells oval, hyphæ produced in liquid medium. Gram positive, not acid fast. No ascospores could be demonstrated.

Cultivation.—It produces a smooth, creamy white, moist glistening layer on Sabouraud's glucose agar. *Biochemical reactions*—Optimum temperature, 37 degrees C. Indol, not formed. Milk, unchanged. Serum, not digested. Gelatin, not liquified. Fermentation of carbohydrates (see table for fermentation). *Identity*, recorded at time of study as "Monilia" (Castellani).

There was found marked infiltration of both vocal cords with hypertrophic changes in the interarytenoid area. The mucosa presented a wrinkled appearance. The left vocal cord appeared nodular. There was no ulceration. The arytenoids were found moderately swollen (Fig. 8). The appearances suggested a tuberculous process. Chest studies were negative for evidences of tuberculosis. Secretions were negative for tubercle bacilli. The Wassermann was negative.

A second case presenting similar clinical findings was that of a woman, aged 48 years, whose voice had been husky for many years.

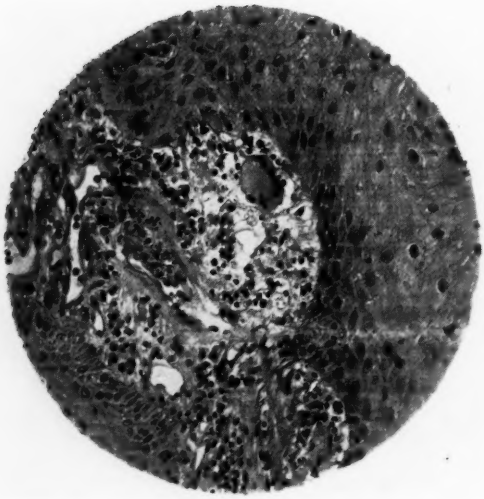

Fig. 7. Photomicrograph of tissue from the patient's larynx, illustrating the granulomatous character of the lesion (x400).

Fig. 8. Drawing of mirror image of larynx. The changes are more marked on the left vocal cord and in the interarytenoid area which present a nodular appearance. The right vocal cord is swollen and presents a wrinkled appearance.

Recently marked hoarseness developed. Studies were negative for tuberculosis and syphilis. Mirror laryngoscopy revealed marked infiltrative changes involving both vocal cords, which presented a nodular appearance. There was a thin film of exudate covering both cords. There was some interarytenoid infiltration. Tissue and secretions were secured from the larynx for study. The tissue showed chronic inflammatory changes with hyperplasia and hyperkeratosis of the mucosa. An anascosporous yeast was found.

Description of the fungus. Morphology.—On Sabouraud's glucose agar the cells are ellipsoid, doubly contoured, with an average measurement of 4 by 2.5 microns. There are occasional large cells, with little structures, up to 7 microns, present in old cultures. The yeast cells bud dichotomously, producing bushy masses of cells, giving the edge of the colony a dendroid appearance (Fig. 9). On beerwort, the majority of the blastospores are lacrimiform, produced in dense parallel masses (Fig. 10). The hyphæ are branched on beerwort agar but little developed on other media. They are terminated by an elongate element or a blastospore. Gram positive, not acid fast. Cells contain globules of fat. No ascospores on carrot, Gorodkova's agar or plaster of paris blocks.

Cultivation.—On Sabouraud's glucose agar, colonies are flat, yellowish white, dull, thin and pasty. The giant colony measures 4 cms. after thirty days, is rather thin, flat, white, smooth, rather dull, with a central eminence and a few shallow, radial striations. There are numerous fine granulations on the surface. The colony is not lobed (Fig. 11).

Beerwort agar, yellow, dull layer, denticulate margins. Beerwort, no pellicle, turbidity clears, granular sediment. Carrot, uniform, thin, white, moist layer. Potato water, no turbidity or pellicle; granular sediment.

Biochemical Reactions.—Pellicle and ring produced on some carbohydrate media (see table for fermentation reactions). Gelatin, beaded growth along stab from which hyphæ project in fan-like arrangement. No liquifaction after sixty days. Indol, not formed. Serum, not digested. Milk, becomes alkaline, not clotted. Optimum temperature, 37 degrees C.

Pathogenesis.—Young rabbits, inoculated intravenously, with 1 cc. of suspension, died after eighteen days. All of the viscera are markedly congested and liver contains numerous discrete white nodules. Microscopically, liver sinusoids filled with blood. The liver cells exhibit cloudy swelling. Masses of branching fungi are present. In these areas there was hemorrhage and necrosis, with only slight leucocytic infiltration.

Laryngeal tissue from the patient is characterized by thickening of the surface epithelium and chronic inflammatory change in the submucosa. The tissue is edematous, contains many new blood vessels, growing connective tissue and a moderate infiltration with leucocytes and round cells. Fungus was recovered from lesion by culture but could not be demonstrated in stained sections.

Identity.—This fungus is believed to belong to the genus Blastodendrion because of the lacrimiform blastospores in beerwort, the parallelism of the cells and the dendroid clusters.

Fig. 9. Photomicrograph of margin of colony on glucose agar, illustrating dendroid clusters of the organism (x80).

Fig. 10. Photomicrograph of margin of a colony on beerwort agar illustrating hyphæ and elongate lacrimiform blastopores in parallel arrangement (x80).

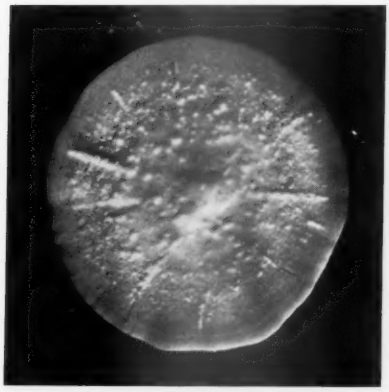

Fig. 11. Photograph of giant colony of fungus, after 40 days. See text for description.

FERMENTATION OF CARBOHYDRATES BASED ON A SERIES OF TEN TRIALS EACH

Carbohydrate 1%	Case 1	Case 2	Case 3	Case 4	Case 5
Dextrose	A & G	A & G	A & G	A & G	A & G
Levulose	A & G	A & G	A & G	A & G	A & G
Maltose	A & G	0	A & G	A?	A?
Galactose	A & G	A & G	A & G	A	0
Sacchrose	A	A & G	A & G	A	0
Mannose	0	A & G	A & G	A & G	0
Inulin	0	A & G	0	0	0
Raffinose	0	A & G	0	0	0
Dextrin	A?	0	0	A	0
Lactose	0	A & G	A	0	0
Mannite	0	0	0	0	0
Dulcite	0	0	0	0	0
Arabinose	0	0	0	0	0
Adonite	0	0	0	0	0
Sorbitol	0	0	0	0	0
Xylose		0	0	A?	
Rhamnose		0	0	0	0
Salicin		0	0	0	0
Trehelose		0	A & G	0	

A, Acid; G, Gas; 0, No change; ?, Doubtful. Blank space, not tried. Medium, Beef extract broth. PH-7, Indicator bromcresol purple.

SUMMARY

In extensive chronic inflammatory lesions of the larynx with ulcerative and exudative changes which, although suggestive of tuberculosis are not characteristic of any definite clinical entity, one should consider fungus infection as a diagnostic possibility. In the first three

cases a tentative diagnosis of tuberculosis had been made. Repeated negative sputum studies for tubercle bacilli and negative pulmonary findings in spite of histologic diagnoses of chronic ulcerative laryngitis, either granulomatous or suggesting tuberculosis, indicated a need for additional investigation. The final diagnosis rested on the results of the mycologic findings together with the above noted findings. Fungi were demonstrated in all of the lesions. They produced definite lesions in rabbits. The positive mycologic findings at repeated studies and the improvement under iodide therapy are conclusive. In the remaining two cases the findings were suggestive from a clinical standpoint. Although fungi were found in both cases, their pathogenicity is inconclusive.

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LXXV

MIXED TUMORS IN THE NOSE AND THROAT*

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TORONTO

The purpose of this paper is to discuss mixed tumors of the salivary gland type occurring in the nose and throat. To avoid encroaching on the field of general surgery, the six cases reviewed are all of the so-called aberrant type—that is, not directly associated with the salivary glands.

No attempt will be made to discuss in detail the controversy of the classification and origin of these tumors, but a short review of the incidence, pathologic structure and clinical course will be attempted. This type of treatment used in the various cases will be given special consideration in an endeavor to reach some conclusion as to that which is most effective.

Incidence.—Mixed tumors of salivary gland type are not by any means a rarity. They occur at all ages and have been reported in a girl of three months[1] and in a man of 73 years.[2] These tumors may be intimately associated with the salivary glands or may arise in aberrant areas, notably the retropharyngeal region, lateral pharyngeal wall, cheeks, lips, base of tongue, palate, lacrimal gland or accessory nasal sinuses. Although the predominating cellular structures may vary in these locations the tumor must be classified in this group. Sonnenschein,[3] in a splendid review of the literature, found about fifty cases with involvement of the soft palate. There are very few cases with involvement of the antrum. We are reporting two cases from the soft palate, one from the antrum and ethmoid, and discussing one from the nasopharynx and two additional ones with involvement of the palate. These latter two were patients of Dr. H. W. Wookey.

After discussing at length the various arguments regarding the origin of these tumors, Ewing[4] concludes that they cannot be classi-

*From the Department of Otolaryngology, University of Toronto.
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Fig. 1. Photograph of tumor in Case No. 2. This was of 20 years' duration, although it had been removed surgically on one occasion.

fied as true teratomas. The present status of our knowledge of their origin he summarizes as follows: 1. The endothelial origin, as advocated by some, has been disproved. 2. No single origin meets all the requirements. Some are distinctly adenomatous, and probably arise from the acini and ducts of the gland in which they are well incorporated. Others are encapsulated or extraglandular and take the form of basal-celled or adenoid cystic epithelioma. These probably arise from misplaced and occasionally embryonal portions of gland tissue. Branchial remnants may possibly be connected with this group. 3. The derivation of mucous tissue and cartilage, by metaplasia, from

gland epithelium, has practically been proven, and there is no need of including them in the originating tissue.

Fraser[5] gives the most likely proof of development from epiblastic, fully formed gland tissue that has undergone some functional change. He experimentally ligated the parotid duct in dogs and produced changes which, by comparison, led him to conclude that the so-called mixed tumors arise from cells of the gland ducts. The cartilage and mucinous material were found to be formed from the epithelial structures by metaplasia.

Pathologic Structure.—The tumors show broad areas or narrow zones of epithelial cells, which surround and fade into masses of mucoid, connective or cartilaginous tissue. The epithelium shows all gradations from adenomatous alveoli derived from the gland acini and ducts, to solid masses or narrow strands of small, flattened, compact epithelium of a basal type. The transformation of gland structures into tumor tissue at times can be fully traced. In the fibrous or mucinous matrix many collagen and elastic fibers may form. Many focal areas of lymphocytic infiltration may be seen in the stroma.

In slowly growing tumors the epithelial elements may almost entirely disappear, leaving almost pure myxoma or chondroma. This may occur in localized areas so that a biopsy report may be quite misleading.

None of these structures show active growth or mitotis that would place them in the classification of malignant tumors. They are, however, potentially malignant and demand long continued postoperative study.

Clinical Course.—In most cases a small quiescent tumor precedes the development of an active growth. Once established, the active growth proceeds slowly, but varies of course with its histologic type. The average known duration of operative cases is about eight years, but Wood[6] reports an inactive mass of 53 years' duration. There may also be an arrest of activity. Martland[7] reports one tumor that after active growth for two years, remained stationary for thirteen years. After surgical removal encapsulated growths, as a rule, do not tend to recur, but they may soon afterwards, or as late as nine years. In the recurrent tumor the structure may remain more constant, but more often it will become increasingly cellular, atypical and develop malignant characteristics.

Undisturbed tumors rarely invade lymph spaces, but after an unsuccessful operation, while recurrences are usually local, the cervical nodes may be progressively involved.

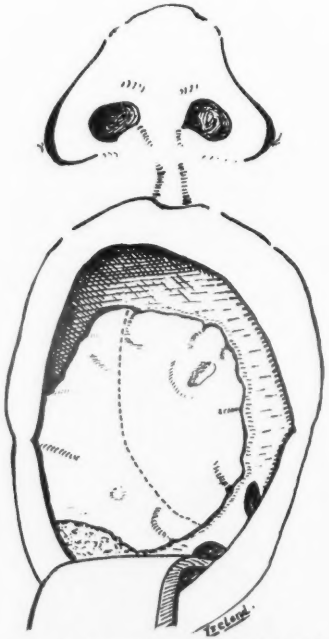

Fig. 2. Method of removal of palatal tumor of Case No. 2. An elliptical incision is made to facilitate the plastic result desired on the soft palate.

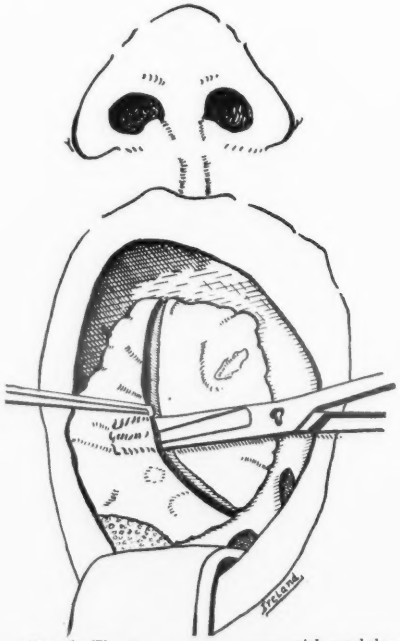

Fig. 3. The tumor mass, even with nodular extensions, can be carefully dissected free by blunt dissection. Care is taken to preserve the nasal portion of the palate.

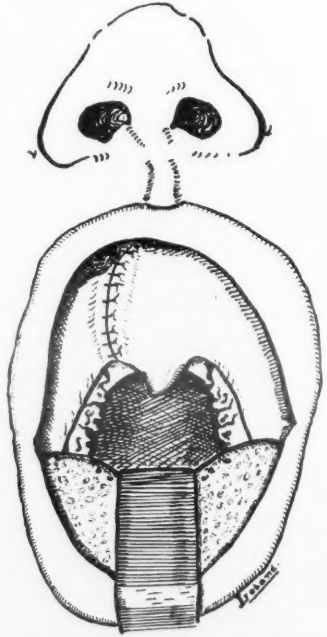

Fig. 4. The incision is closed and the advantage of the lateral elliptical incision is illustrated. There will be very little deformity of the palate or narrowing of the nasopharynx.

REPORT OF CASES

CASE 1. Diagnosis, Mixed Tumor of Palate, Salivary Gland Type.—S. P., age 59, admitted April 4, 1934. Birthplace, Russia.

Chief complaint: Mild sore throat of two weeks' duration.

Present illness: The patient stated that he did not remember when swelling in mouth began. He had no discomfort until two weeks previous, when he had consulted his own doctor for a mild sore throat and was referred into hospital. Remainder of the history and general physical examination were negative.

Special examination: There was a mass the size of a small lemon in the soft palate on the left. This filled the left side of the nasopharynx and displaced the uvula, along with the tonsil on this side, to the right. The appearance was very much that of a peritonsillar abscess. Palpation showed no ulceration and no enlargement of the cervical glands.

Laboratory findings: Urine and blood normal. Wassermann negative.

Pathologic diagnosis (biopsy specimen): Mixed tumor of salivary gland type. (Dr. McGregor and Dr. Robinson.)

Treatment, radio-therapy: This patient was referred to Dr. Richards at the Radium Institute, where a course of teleradium therapy with a 4½ gm. bomb was given. A course of 42,000 mg.h. was given over a three-week period. There was a good skin reaction with epithelitis of mucosal surface but no decrease in the size of the tumor mass, although some central softening was found.

Patient was allowed to go home and admitted a month later with a possibility of insertion of radium needles. The reaction had subsided but the tumor showed marked softening and no needles were inserted. A second course of 60,000 mg. hrs. by a cross firing method with teleradium was given on this admission.

The third admission was on September 25, 1934, four months after the last radium treatment. The mass was about the same size and apparently had not been affected by the treatments. Surgical removal was considered advisable.

Operation, intracheal ether: A preliminary tracheotomy was done and an attempt made to dissect out the tumor in toto. This was found impossible due to varying scarred and softened areas believed to be due to radium therapy. This mass was removed piecemeal and the palate incision closed. No perforation was obtained. Tracheotomy tube was removed at the end of operation and the incision was pulled together by a dressing.

Progress: Two years later patient was entirely free from symptoms and there was no evidence of recurrence of tumor in the palate.

Summary: This tumor was treated first by radium in very intensive doses with a good reaction, but the results showed no apparent regression of the tumor mass. Subsequent surgery was probably made more difficult by the previous radiation.

CASE 2. Diagnosis, Mixed Tumor of Palate, Salivary Gland Type.—E. W., age 51, admitted February 28, 1935. Birthplace, Poland. Chief complaint: Tumor in roof of mouth, twenty years' duration.

Present illness: In 1914 patient first noticed a swelling in the roof of the mouth. This gradually increased in size until it was removed surgically in 1920. A few years later she could feel a recurrence of the tumor, that on admission was sufficiently large to cause difficulty in swallowing and obstruction to breathing.

Personal history: Born in Poland and emigrated to Canada twenty-four years ago.

Physical examination: Patient showed a degenerative myocarditis with hypertension. Her blood pressure was systolic 210, diastolic 120.

Laboratory findings: Urine negative, blood examination normal, Wassermann negative.

Special examination: There was a smoothly nodular mass in the right side of the soft palate about the size of a lemon. It was more on the right side and extending to the right supratonsillar fossa but not into the lateral pharyngeal wall. The uvula was pushed to the left but the mass extended well past the midline. The hard palate was not involved and digital examination of the nasopharynx revealed, when the tumor was displaced downward, that the opening into both choanæ was still present. The tumor on palpation seemed encapsulated, was freely movable, rubbery in consistency, but with several firm nodules in the deep structure. There was no ulceration and the glands of the neck were not palpable.

Biopsy: The mucous membrane was split and a section taken of deep structure, including one of the nodules present.

Pathologic report: Mixed tumor of salivary gland type. (Dr. McGregor, Dr. Robinson.)

Operation (Dr. Goldsmith), intracheal ether anesthesia: In the Rose position the mass was exposed by an elliptical incision and dissected out. There was a capsule present but with nodular extensions through it. The whole tumor was removed and palatal surface of mucous membrane was sutured.

Progress: Palate healed well, but the right tonsil still remained more prominent than the left, so one month later patient was readmitted and tonsils removed, with the anterior pillar and supratonsillar fold on the right.

Patient is to receive a postoperative course of radium therapy by means of teleradium bomb, as advised by Dr. Richards of the Radium Institute.

Summary: Here is a patient with a twenty-year history treated by surgery fifteen years before with no radium therapy. Growth had recurred slowly until it was causing obstruction to breathing and swallowing. Thorough surgical removal has been attempted, to be supplemented by a course of radium therapy to prevent recurrence in any remaining active elements. Patient has an intact palate with normal movements that probably would not be the result had diathermy been used in the removal of the tumor.

CASE 3. Diagnosis, Mixed Tumor, Salivary Gland Type Involving Antrum, Ethmoid and Septum.—W. R., aged 65. Admitted December 21, 1933.

History: Patient was first seen on December 12, 1933, with a complaint of gradually increasing right nasal obstruction, accompanied by nasal discharge. These symptoms were first noticed seven months previously. He also gave a history of epistaxis on six occasions during the last year and a half, but these were not sufficiently severe to cause worry and he did not consult his doctor regarding them.

Examination: The right nostril was found to be completely blocked by a large, very vascular, cystic type of mass. This eroded the septum and extended into the antrum, with the naso-antral wall apparently largely destroyed. The tumor mass could be seen in the right posterior choanal arch by posterior rhinoscopy.

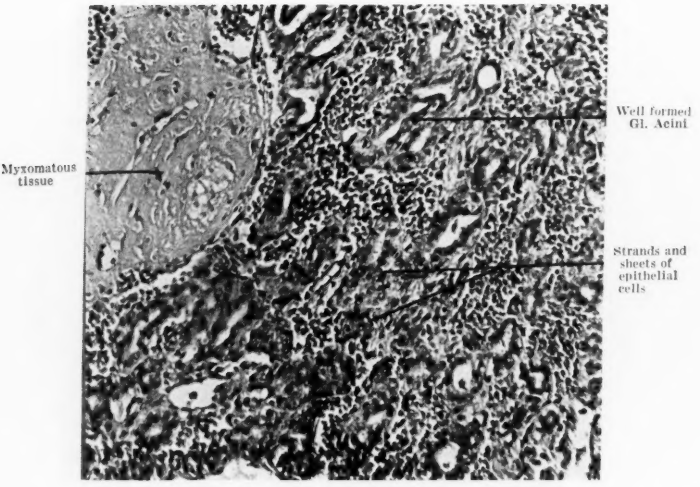

Fig. 5. Mixed Tumor of Antrum (Case No. 3). Tissue from antrum showed considerable variation in structure. In some areas the bulk of the tissue was composed largely of acinous structures while in other areas there is a large amount of myxomatous type of tissue present.

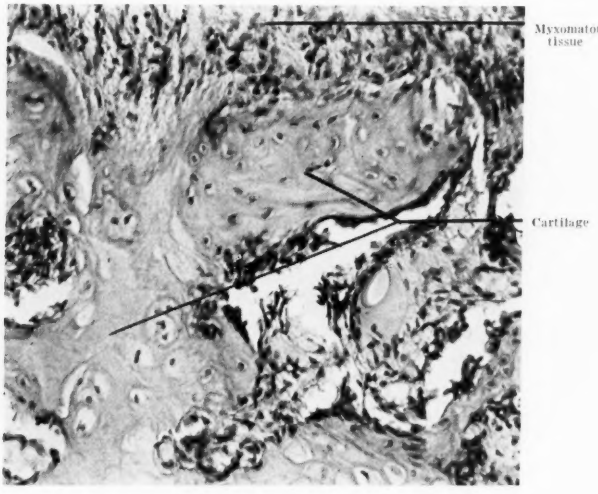

Fig. 6. Mixed Tumor of Palate. Showing islands of cartilage, which would lead to an erroneous diagnosis of chondroma if biopsy showed only such an area.

X-ray films were made of the accessory nasal sinuses. The radiologist gave the following report: "The frontals appear normal in size and extent and are apparently clear. The anterior ethmoids are well formed, and the right is completely obscured and the left appears clear. The maxillary antra are normal in size and extent. The right is densely thickened throughout and the same type of density is present on the whole right side of the nose and apparently bulges through to the left, both above and below. We can detect no definite evidence of bone destruction in the wall of the antrum, but the findings are those commonly found with a malignant growth involving the antrum and the nose, causing complete obstruction on this side and bulging slightly through to the left. The right antrum appears normal. The sphenoids and posterior ethmoids are slightly cloudy on the right side, apparently due to the nasal obstruction which is present."

Biopsy: Mixed tumor salivary gland type (Dr. Robinson and Dr. McGregor).

Operation, December 12, 1933: The canine fossa was exposed and a small opening in the front wall of the antrum was found with an erosion of the tumor through it. The antrum was completely filled with a vascular, boggy mass that extended into the nose, filling the ethmoid region and with apparent involvement of the sphenoid.

The entire mass was removed, together with the remaining naso-antral wall, inferior and middle turbinates, the remaining ethmoid region and the front wall of the sphenoid. There was found an erosion by the tumor mass of the os planum of about one inch in length, exposing the orbital tissues.

An area on the septum was first thought to be a traumatic erosion, but the cartilage and mucosa in this area were removed also for pathologic examination.

Pathologic diagnosis: A diagnosis of mixed tumor was confirmed, with involvement of antrum, ethmoid and septum. The mucosa from the sphenoid showed thickened membrane only.

Radiation, December 23, 1933: Into the antrum were placed four 10 mg. platinum tubes for twelve hours, giving a total dosage of 480 mg. hours.

Results: Patient developed marked reaction from the radiation with a great amount of swelling of the eye and face. There was proptosis of the eye, and the conjunctiva was very edematous and corneal sloughing was feared. Necrosis involved the floor of the orbit, the bone being black in color. As the malignant nature of the mass was overestimated, it was feared that an orbital extension had been started. But the eye gradually receded and all inflammatory reaction subsided after several weeks, it is probable that the process was due to the radiation. Diplopia, however, remained, owing to the disturbance of the orbital floor. The vision was not affected. The patient became dissatisfied and left the hospital against advice to take a course of popular "cancer serum" treatment. In due time the radium reaction subsided and the patient was relieved of symptoms, and of course he attributed the cure to the cancer serum.

This man has remained free from recurrence, as one can observe through the permanent antra-buccal opening.

CASE 4. Diagnosis, Mixed Tumor of the Soft Palate, Salivary Gland Type.—C. A., age 33. Admitted November 1, 1934, a patient of Dr. H. Wookey.

(This case is reported to note treatment.)

Gross appearance: A swelling was present in the roof of the mouth that extended forward to about the middle of the hard palate and back to the posterior limits of the soft palate. The surface was raised and somewhat irregular but covered by intact epithelium.

Diagnosis, biopsy: Mixed tumor showing early malignant change (Dr. W. L. Robinson), November 1, 1934.

Radiation: Three 2 mg. and one 5 mg. platinum needles were inserted in the mass for 120 hours, giving a total dosage of 1320 mg. hours.

Progress, February 1, 1935 (three months later): Patient had a fistula through into the nose with thickened unhealthy margins.

February 1, 1935, biopsy: Still shows mixed tumor but without the evidence of malignant changes previously noted.

April 16, 1935, repeat radiation: Five 1 mg. platinum needles were inserted in a radial manner extending out from the margin of the ulcer and left for 120 hours, giving a dosage of 600 hours.

September 20, 1935: Patient was unable to wear the dental plate made to close fistula. The opening was larger than before the last radiation and the margins were still thick, although active disease did not seem evident. No attempt at plastic closure was thought advisable.

CASE 5. Diagnosis, mixed tumor of the palate, salivary gland type.—L. B., age 48. Admitted May 12, 1934, a patient of Dr. H. Wookey.

History: Swelling in the roof of the mouth, on the right side, of two months' duration.

Examination: A swelling was seen in the soft palate, on the right side, about the size of an olive. This presented a number of grayish colored erosions from which a somewhat caseous material could be expressed on pressure.

Biopsy: Mixed tumor, salivary gland type, that was very similar to parotid gland tissue.

Treatment: Six 1 mg. platinum needles were inserted into tumor and left for 120 hours, giving a total dosage of 720 mg. hours.

Results: In January of this year this patient was seen and apparently has an excellent result. There is only a small scarred area in the palate and no evidence of recurrence can be seen. This, of course, is only after two years.

CASE 6. Diagnosis, mixed tumor, salivary gland type, of nasopharynx.—W. H., age 47. Admitted March 9, 1933 (patient of Dr. G. McGregor).

History: This patient was perfectly free from symptoms of any sort. The presence of a tumor was noticed accidentally by his wife, while looking in his mouth.

Examination: There was a smooth rounded tumor mass giving a fullness to the soft palate and showing below the level of the uvula. It was about the size of a large walnut and rubbery in consistency. No glands were palpable in the neck.

Treatment: Tumor mass was dissected out from the capsule covering it and the wall of the nasopharynx sutured in place. Patient received a prophylactic postoperative radiation series.

Results: At the end of three years there has been no evidence of recurrence.

SUMMARY

The treatment in these various cases has been reported in detail for purposes of consideration. As you will see, our experience with radiation therapy alone in this small series has not given satisfactory results. In the three cases involving the palate, treated primarily with radiation, only one was apparently successful. The one treated with massive dosage by the 4 gm. teleradium bomb seemed little affected, and eventually surgery was made more difficult. One other case treated by needles had an unsatisfactory result, even after a repeated course of therapy. There was an opening into the nose present and indurated tissue, still suspicious of tumor, after the lapse of a year. The case successfully treated was a small tumor, of apparently short duration, with well differentiated salivary gland tissue.

The last palatal case was treated first with surgery, presenting no difficulties of dissection and giving an apparently good result. The one with involvement of the accessory sinuses and that of the nasopharynx were treated by surgical removal and postoperative radiation with good results.

Mixed tumors of this type do tend to recur, and the length of time elapsed is not sufficient to speak of cures, but the immediate result of thorough surgical removal seems to contraindicate any suggestion of radiation as a first consideration. A prophylactic, short, postoperative course of treatment may be beneficial in obviating recurrences, especially of the cell types, with malignant tendencies.

CONCLUSIONS

1. Six cases of aberrant mixed tumors of salivary gland type have been reported.

2. The general concensus of opinion is that these tumors are not true teratomas. Those closely associated with the glands proper probably arise from the gland ducts, and the aberrant type from embryonal rests. Cartilage and myxomatous tissue can be developed by metaplasia and their mesodermal origin is not considered essential.

3. Tumors of this type involving the accessory sinuses are rare. One such case has been reported.

4. The treatment by complete surgical removal was the most satisfactory measure in this series.

5. Radiation as a primary treatment should not be considered. Prophylactic postoperative radiation may have some usefulness.

6. Recurrence of the growth is frequent, and no attempt to report any of the cases as cures has been made.

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LXXVI

THE VALUE OF ROENTGENOGRAPHY OF THE NECK WITH SPECIAL REFERENCE TO ITS USE IN THE DIAGNOSIS AND TREATMENT OF LARYNGEAL AND TRACHEAL OBSTRUCTION*

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Very soon after Roentgen's discovery, the first paper on the x-ray study of the laryngeal cartilages was read (1896) by Scheier,[1] who later published a study of the speech mechanism (1909) and a study of the swallowing act (1911). Fränkel[2] also studied the ossification of the larynx at about this time. Thost[3] published his studies on the normal and diseased larynx in 1913.

In this country, Iglauer[4] seems to have been the pioneer writer on the value of roentgenography in the diagnosis of disease of the larynx and trachea. He published a paper on the subject in the Journal of the American Medical Association in 1914.

For over ten years subsequent to this time, very few references are found in the literature, but Pfahler[5] contributed an excellent study of the thyroid gland (1921). In 1927, Mosher,[6] in collaboration with Macmillan, made some very interesting "x-ray studies of the movements of the tongue, epiglottis, and hyoid bone in swallowing." In the next year, Hickey[7] discussed the radiography of the larynx in his Caldwell lecture, and an interesting paper by Brown and Reineke,[8] on the roentgenologic study of the neck, appeared.

Chevalier Jackson has had great faith in roentgenology since its earliest beginnings, and for many years "neck, chest and swallowing function" has been almost a routine request in the preliminary study of patients in his clinics. It is to the fact that Jackson always insisted on roentgenologic study of the neck that Hay attributes the interest in this subject which led him to prepare his well known monographic atlas[9] in 1929. Many of the cases studied by Hay were referred by the Bronchoscopic Clinic.

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*Presented before the fifty-eighth annual meeting of the American Laryngological Association, Detroit, May 27, 1936.

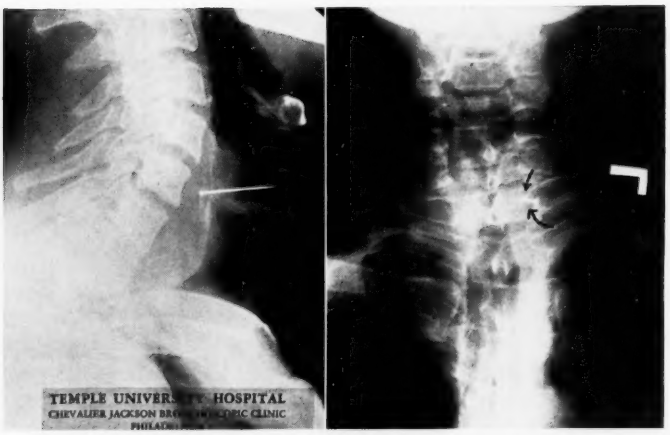
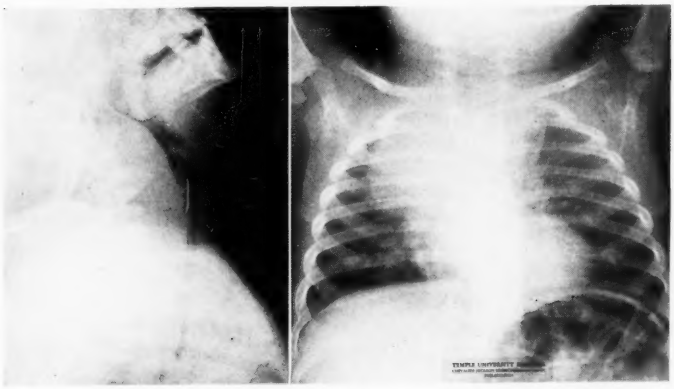


Fig. 1. Common pin embedded in soft tissues of interior of larynx, lying along internal surface of left wing of thyroid cartilage. This foreign body was removed by "window resection" (C. J.) of the cartilage.



A B

Fig. 2. Roentgen study is of great aid in studying retropharyngeal abscess, and the chest should be included in this study because of the frequency of mediastinal involvement.

(A) Lateral view of neck showing shadow of large retropharyngeal abscess in a child of 13 mos. of age.

(B) Film of chest taken at same time, showing extension of abscess into superior mediastinum. Uneventful recovery followed external surgical drainage (W. Wayne Babcock).

Fig. 3. Note shadow of markedly thickened epiglottis. The patient had an epiglottic abscess giving pain on swallowing, of two days' duration.

It was the writer's privilege, in 1930, to study a series of cases of laryngeal disease with Dr. Henry K. Pancoast, and to discuss them with him at the meeting of the American Medical Association in Detroit.[10a, 11c] Ever since that time the writer has been much interested in the roentgenologic study of the neck and has asked for the roentgenologist's help in a great many different kinds of case. The present paper is based upon a paper on "The Value of Roentgenography of the Neck to the Laryngologist," read by invitation before the New England Roentgen Ray Society in Boston several years ago but not published. It is the writer's intention to review the various uses of roentgenography of the neck, referring briefly to numerous illustrative cases. Particular attention will be paid to the uses of roentgenography as an aid in the diagnosis of obstructive diseases of the larynx and trachea, and in the study of the size, shape and position of tracheotomy tubes and laryngostomy apparatus.

Foreign Body.—Foreign bodies in the larynx, trachea or cervical esophagus, if opaque, are well shown in the lateral roentgen view, and it is easy to demonstrate whether they are in the air or in the food passages, because the larynx and trachea are clearly seen to lie anterior to the esophagus. Another helpful point is that foreign bodies

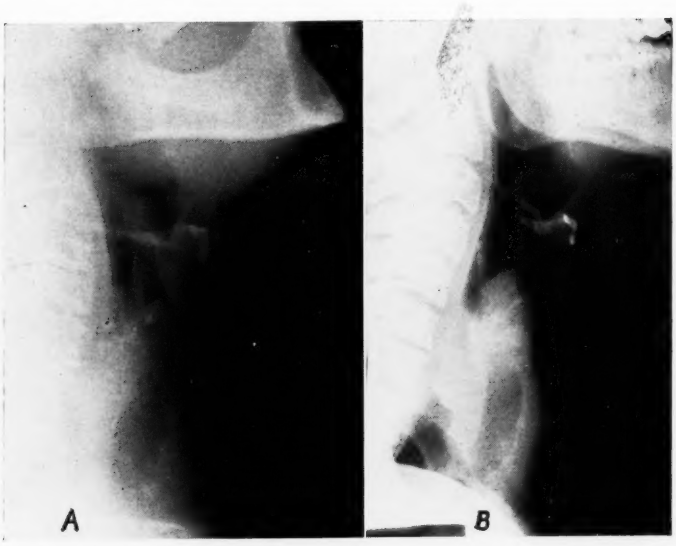


Fig. 4. Two cases of scleroma of the trachea showing similar irregularities of the tracheal outline.

(A) Subglottic and tracheal stenosis in an Austrian woman, 55 years of age, due to rhinoscleroma.

(B) Tracheal stenosis in young man born in Pennsylvania, in whom a definite diagnosis of scleroma was made by biopsy.

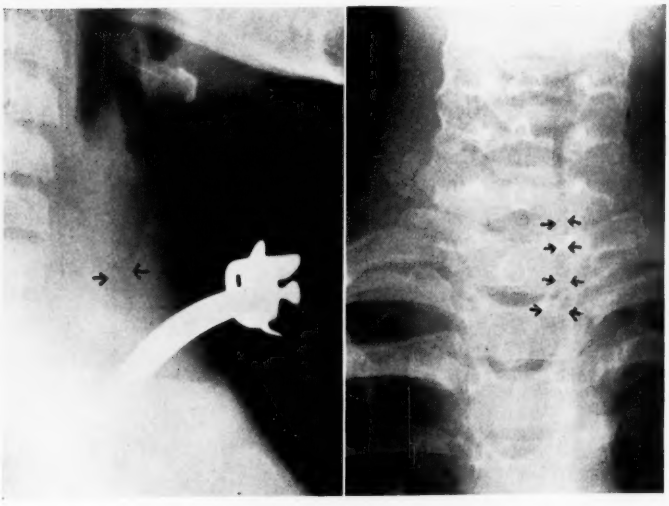


Fig. 5. Compression stenosis of trachea in a child 10 years of age, due to thyroid tumor. Note particularly anteroposterior view.

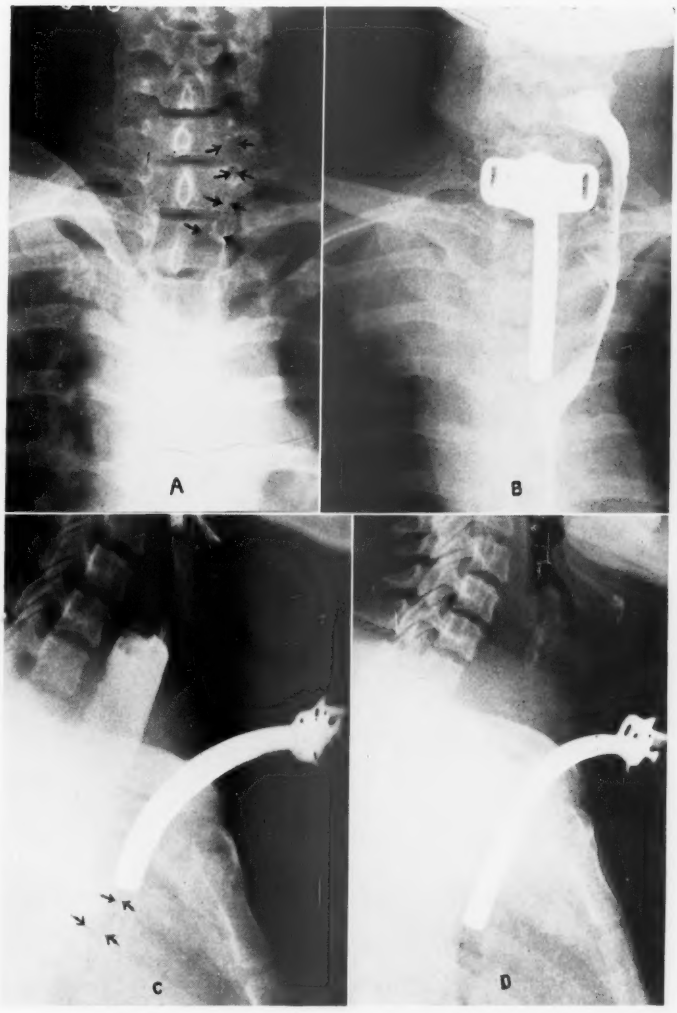


Fig. 6. Serial roentgen studies are of great value in the treatment of tracheal stenosis due to Hodgkin's disease or mediastinal neoplasm.

(A) Anteroposterior view showing stenosis of trachea by mass involving the right lobe of the thyroid gland in a case of Hodgkin's disease.

(B) Film of same patient a number of months later, showing great increase in mass. A cane-shaped tracheotomic cannula is in place and the opaque mixture demonstrates a marked deviation of the esophagus.

(C) Lateral view showing tracheal stenosis just below distal end of ordinary tracheotomic cannula.

(D) Lateral view showing cane-shaped cannula which was substituted for ordinary cannula shown in (C) in order to reach below level of stenosis.

in the larynx and trachea almost always lie in the sagittal plane, while those in the cervical esophagus lie in the coronal plane.[12]

One important item in differential diagnosis which has already been emphasized by Chamberlain and Young,[13] and also by Taylor,[14] is the fact that small spots of ossification in the laryngeal cartilages may be mistaken for small bones in the esophagus. As pointed out by the writer in discussing Taylor's paper, bones are more often lodged just below the level of the larynx and hence below the level of localized ossification, but they do also, not infrequently, lodge just behind the cricoid cartilage, and in such cases it may be quite difficult to differentiate them roentgenologically from a plaque of ossification in the lamina of the cricoid. Occasionally foreign bodies become embedded in the soft tissues, in which case roentgen localization is invaluable. (Fig. 1).

Retropharyngeal Abscess.—Roentgen examination is one of the most reliable means of detecting and following the progress of retropharyngeal abscess, and it is important to keep the chest and especially the mediastinum under roentgen observation, because such lesions may extend insidiously into this region. (Fig. 2.)

Disease of the Hypopharynx and Cervical Esophagus.—Benign strictures of the cervical esophagus are fairly common in elderly women, and it is important to differentiate them from carcinomas. Pulsion diverticula of the hypopharynx are in most cases quite typical in their roentgen appearance, but occasionally an erroneous diagnosis is made because pouching above a stricture may simulate a true pulsion diverticulum of the hypopharynx.

Laryngeal Edema, Inflammatory Stenosis of the Larynx and Trachea, Tuberculosis and Syphilis, Scleroma, Compressive Stenosis of the Trachea.—Laryngeal edema may be studied and followed very easily by the roentgenologist. We have found his aid of great value in determining conditions present in cases of acute infective laryngotracheitis, laryngeal diphtheria, edema secondary to "habit spasm," and edema due to foreign body. Edema or abscess of the epiglottis is especially well visualized. (Fig. 3.) Tuberculous and syphilitic laryngeal lesions very often present roentgen appearances that are instructive in connection with diagnosis, treatment and prognosis, but of course a definite differential diagnosis of such conditions cannot be made from the roentgen study alone. Scleroma may produce subglottic or tracheal stenosis, and such a lesion lends itself readily to roentgenographic study. In our two most recent cases the roentgen resemblance was striking. (Fig. 4.) Tracheal stenosis may also be produced by compression, as a result of enlargement of the thy-

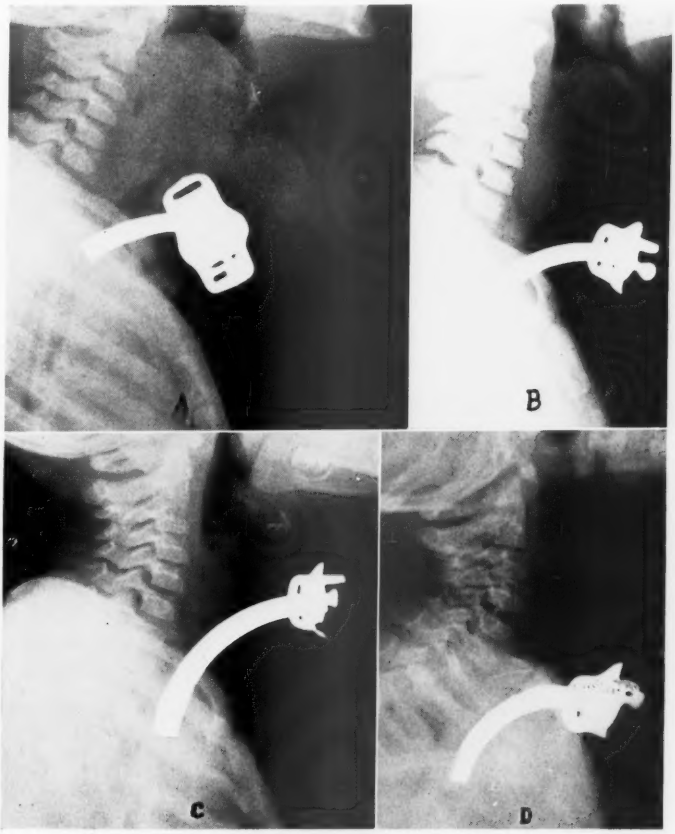


Fig. 7. While small benign tumors of the vocal cord can generally be visualized, it is in the larger tumors that roentgen study is of greatest practical value.

(A) View showing large tumor of neck in child 3 years of age.

(B) After removal of external portion of tumor (fibrolipoma) this roentgenogram showed a large intralaryngeal mass remaining. Direct laryngoscopic inspection showed only the upper surface of the mass and gave a less accurate idea of its form and extent.

(C) After removal of the intralaryngeal portion of the tumor through a thyrotomy incision the cannula was temporarily inserted rather high.

(D) View taken after removal of cannula from thyrotomy incision and insertion low in the trachea. Note that there is little evidence of airway in the larynx and upper trachea. After subsidence of inflammatory reaction a series of direct laryngoscopic dilatations were instituted in order to re-establish the airway.

Fig. 8. Large extrinsic carcinoma of larynx involving chiefly its posterior portion. Note that outline of epiglottis and anterior portion of ventricles is normal.

Fig. 9. Extensive carcinoma of hypopharynx and cervical esophagus. Note destruction of body of cervical vertebra.

roid gland (Fig. 5), of the thymus, or of the mediastinal lymphatic glands, as in Hodgkin's disease. (Fig. 6.) In studying laryngeal and tracheal stenosis the anteroposterior view is sometimes of value as well as the lateral.

Benign Growths.—Even very small benign growths on the vocal cords may often be seen as small defects in the shadow of the laryngeal ventricle, but it is especially in the case of larger tumors that the roentgen study is helpful. By indirect and even by direct examination it is difficult to determine the true size, location and attachments of such a tumor. It is often impossible to determine the relations of its deeper portions.

In one of our cases, following the removal of a large fibrolipoma, it was seen on roentgen study that an intralaryngeal portion remained. (Fig. 7.) This was removed, after splitting the larynx, and subsequent studies have shown the progress made as treatment has been carried on with a view to re-establishment of an airway and ultimate decannulation.

Carcinoma.—As in the case of syphilis and tuberculosis, it is impossible to make a certain diagnosis of carcinoma of the larynx from fluoroscopic or roentgenographic study, but the roentgenologists have found it possible to tell a great deal about the extent of a malignant tumor of the larynx or hypopharynx (Figs. 8 and 9), and they certainly obtain great aid from comparative roentgenographic studies in the neck in following the progress of patients under treatment by irradiation.[15 16 17 18]

It may at first seem to be of purely academic interest for the roentgenologist to study the larynx, but in at least one of our patients roentgen study of the neck was of life-saving value. The patient had been sent to the Roentgen Ray Department by an internist for roentgen study of the chest in order to determine, if possible, the cause of a wheeze. The roentgenologist (Dr. Edward Chamberlain), according to his usual custom, fluoroscoped the patient before taking films, and looked closely at the neck before looking at the chest. He was surprised to find a flopping shadow which at once suggested a pedunculated tumor of the larynx as the cause of the patient's symptoms (Fig. 10). Before this patient left the department he went into the dressing room, and as Dr. Chamberlain was passing by the door of the dressing room he heard a thud against the door. Thinking the patient might be in trouble, he opened the door with a master key and found him cyanotic on the floor, fighting for air. Remembering the fluoroscopic picture, he quickly threw the head back, with the result that dyspnea was relieved and asphyxia averted. On mirror

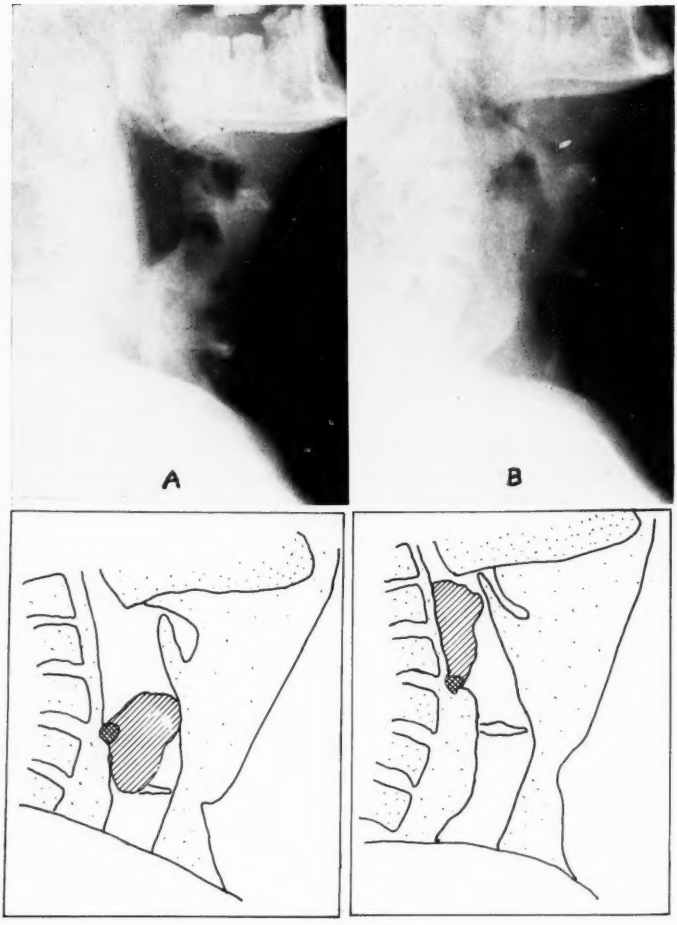


Fig. 10. Large pedunculated carcinoma of the larynx discovered on fluoroscopy of the neck of a patient referred to the x-ray department for roentgen study of the chest to determine the cause of wheezing. Note that mass obstructs the glottis on inspiration or on throwing the head forward, while on expiration or on throwing the head back, the airway is free. (See text.)

Fig. 11. Intratracheal carcinoma of thyroid in a young colored woman. The diagnosis was confirmed by tracheoscopy but the roentgen study gives an accurate idea of the location and form of the tumor.

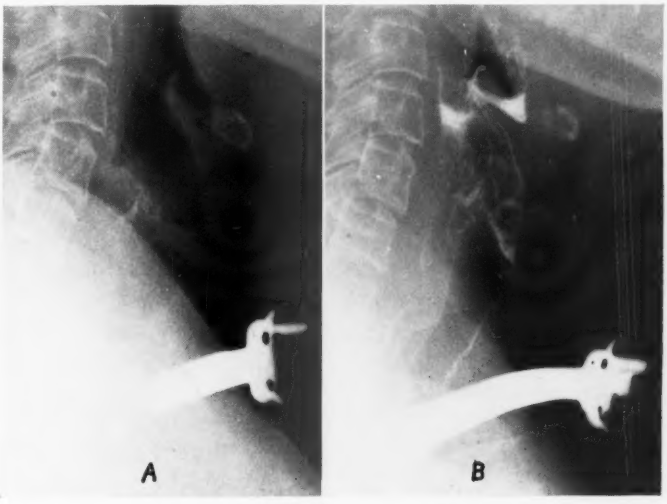


Fig. 12. Laryngography by the use of lipiodol and other opaque contrast media is of great value as a supplementary study.
(A) Ordinary roentgenogram of neck, showing what appears to be a normal epiglottis and normal laryngeal ventricle.
(B) Tumor mass in larynx outlined with opaque mixture.

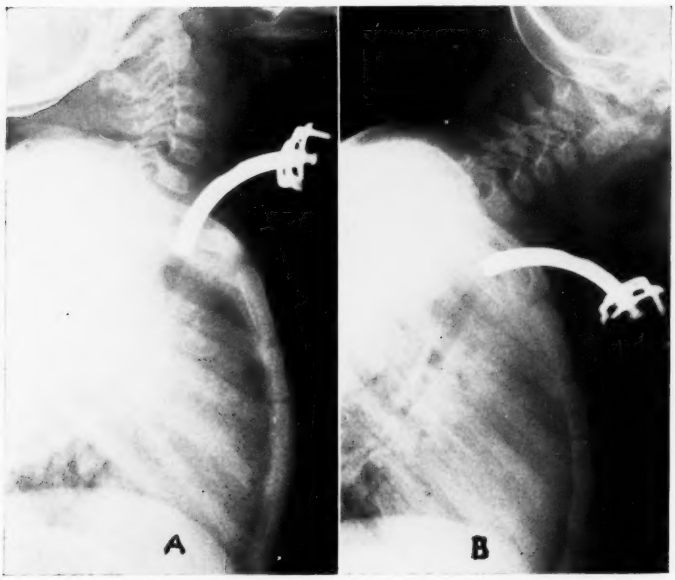


Fig. 13. It is very important for a tracheotomic cannula to be of proper curve and proper length. Roentgen study will show clearly the relation of the distal extremity of the tube to the anterior and posterior tracheal walls, but it is important that the study be made with the head and neck in the average normal position.

(A) When the head is thrown back the cannula impinges on the anterior tracheal wall.

(B) When the head is thrown forward the distal end of the cannula impinges on the posterior tracheal wall.

It is also interesting to note in these films, that the tube is a fenestrated one and that in (A) the fenestrum seems to be in line with the airway, while in (B) it is distinctly in contact only with soft tissues. (Compare Fig. 16.)

examination, the writer found a large pedunculated tumor attached to the left aryepiglottic fold, which was subsequently removed with "basket punch" forceps under direct laryngoscopy. Histologic study showed the lesion to be a squamous cell carcinoma, and a course of postoperative deep roentgen therapy was given by the Coutard technic. To date there has been no recurrence (over three years).

Carcinoma of the trachea and intratracheal thyroid tumors (Fig. 11) are beautifully shown in roentgenograms, and this method constitutes an important part of the preliminary diagnostic study of such cases, regardless of what treatment is to be used.

Laryngography.—In order to obtain better outlining of the laryngeal structures a small amount of lipiodol or other opaque mixture may be instilled, after cocainization, if desired (Fig. 12). This method will sometimes demonstrate structures not clearly shown in plain films.

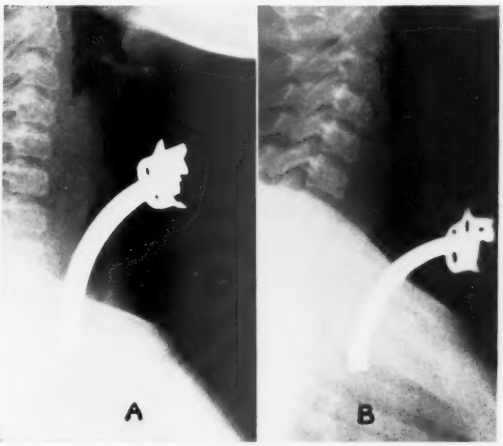


Fig. 14. Laryngeal and tracheal stenosis is never satisfactorily treated unless the tracheotomic cannula is well below the level of stenosis.
(A) High tracheotomy. Note stenosis immediately above the tube.
(B) Same patient after low tracheotomy preliminary to the institution of a series of direct laryngoscopic dilatations. Note that the distal extremity of the tracheotomic cannula does not impinge on either the anterior or the posterior tracheal wall.

Tracheotomy Tubes.—Much has been written about the ill effects of high tracheotomy, and there is no question but that the first step in treating laryngeal stenosis is to determine roentgenographically the relation of the tube to the larynx, and the relation of the distal end of the tube to the anterior and posterior tracheal walls when the head is in the normal average position. (Fig. 13.) If the tube is found to be in contact with the cricoid, or inserted through the cricothyroid membrane, a low tracheotomy should be done, as the first step in treatment. (Figs. 14 and 15.) In some patients it will be found that a special curve or, more often, a special length of tube, will be required to prevent impingement on the anterior or posterior tracheal walls. Tucker has advised that a shorter curve be used in patients who are obliged to wear tubes for a long period of time, because in such patients there is no swelling of the tissues and very little dressing is worn. Roentgen study shows conclusively that fenestrated tubes are objectionable, because the fenestrum is almost always in contact with tissues rather than in line with the airway, and is hence traumatizing as well as useless. (Fig. 16.) In most cases there is a sufficient by-pass of air alongside the tube if the tube is of proper size.

Laryngoscopy Apparatus.—As in the case of tracheotomic cannulæ, roentgenography is of great aid in the placement of laryngos-

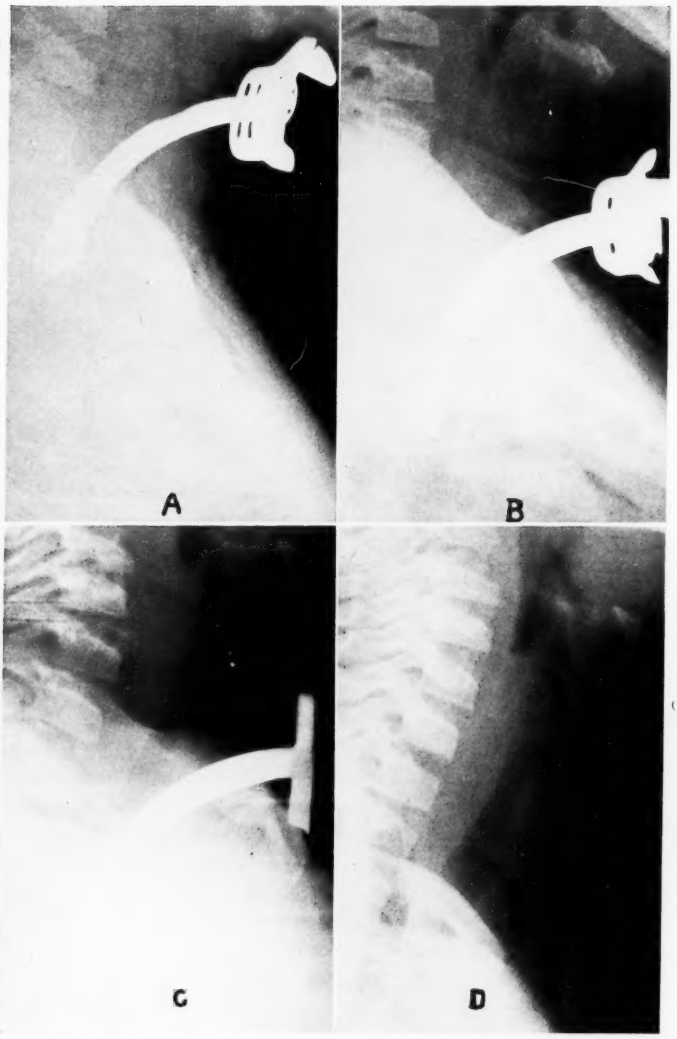


Fig. 15. Laryngotomy may be permissible as an emergency measure in threatening asphyxia, but it should be immediately followed by low tracheotomy.

(A) View of neck showing tracheotomy tube inserted through emergency laryngotomy incision.

(B) View of neck after bronchoscopy for removal of foreign body (nut kernel) from trachea, and low tracheotomy. Note excessive interstitial emphysema and complete loss of laryngeal and upper tracheal outlines.

(C) View several weeks later showing restoration of normal appearance of larynx and trachea. The tracheotomic cannula has been replaced by a blind plug preliminary to decannulation.

(D) After decannulation, the trachea still shows slight irregularity, but the laryngeal structures have resumed normal form and the airway is adequate.

Fig. 16. Note that fenestration in this tube is not in line with the airway, but with the soft tissues anterior to it. Fenestrations too often cause irritation of the tracheal cartilages and soft tissues, as in this case, as well as failing to serve their intended purpose of permitting more air to pass through the mouth. It is preferable to use an ordinary cannula of such a size as to permit by-passage of air. Note also that this tube is of too short a radius of curvature, with the result that its extremity impinges on the anterior tracheal wall. (Compare Fig. 13.)

tomy apparatus, whether the apparatus used is a simple Jackson laryngostomy tube or some more complicated device. When rubber plugs are fixed in place by silver wire according to the method of Schmiegelow, it is necessary to determine the length and relations of the plug as well as the position of the wire. In the case of the "core moulds" recently described by Chevalier Jackson, roentgenograms are always taken to verify the size, length and position of the "mould." (Fig. 17.)

CONCLUSIONS

The roentgenologic study of the neck, by both fluoroscopy and films, with and without opaque mixtures, is often of very great aid to the laryngologist, though, as Ross Golden[19] has said, it will probably never be quite so important as roentgen study of the gastro-intestinal tract and the lungs:

1. Bones in the cervical esophagus can be visualized in the great majority of cases, but care must be exercised not to mistake isolated bits of ossification in the laryngeal cartilages for foreign body, and vice versa. While bones generally lodge at a slightly lower level, not infrequently they are found just behind the cricoid.

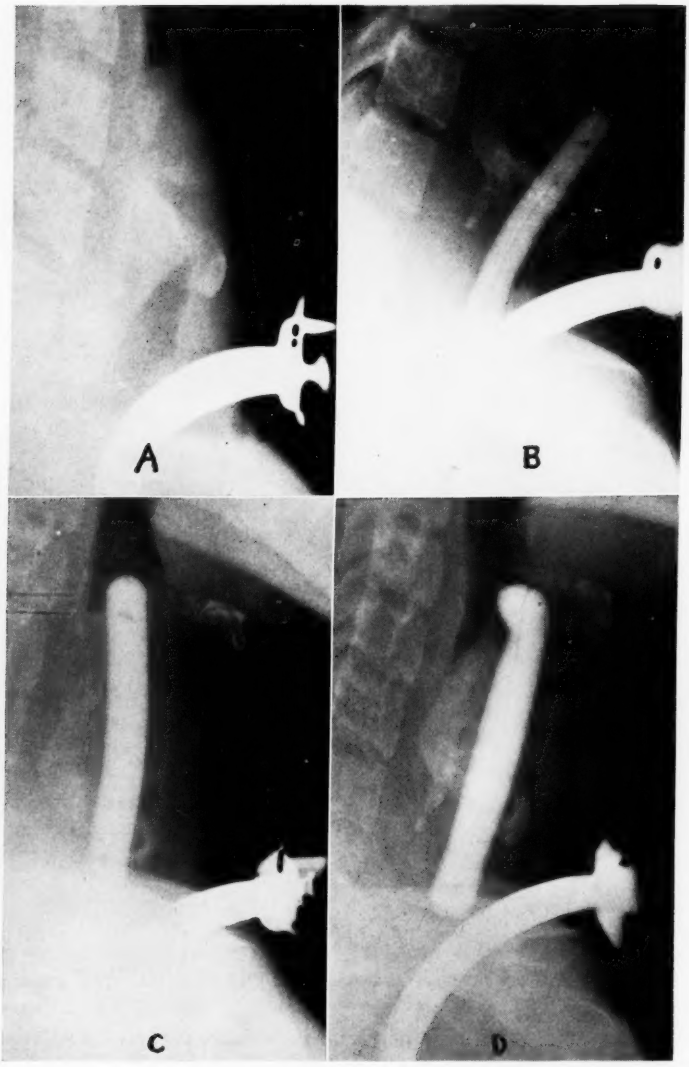


Fig. 17. Serial roentgen studies are of great value in the treatment of laryngeal stenosis with various kinds of laryngostomy apparatus.

(A) Laryngeal stenosis due to perichondritis following an attack of infectious mononucleosis.

(B) View showing solid rubber plug inserted at laryngostomy.

(C) Longer plug inserted several days later. Note the relation of the upper end of the plug with the epiglottis.

(D) New type "core mould" (No. 36) in place. (Described by Chevalier Jackson at the meeting of the American Laryngological, Rhinological and Otological Society, in Denver, 1936.)

2. Foreign bodies in the larynx lie in the sagittal plane; those in the esophagus lie in the coronal plane. Consideration of this point will generally localize the foreign body, but in every case a lateral roentgenogram should be made. The lateral view will show a foreign body in the esophagus lying posterior to the trachea and a foreign body in the larynx or trachea lying anteriorly.

3. Retropharyngeal abscess may be diagnosed early by widening of the retropharyngeal space, as seen in a lateral film, and its course of development can be followed by serial roentgen studies.

4. Edema and other manifestations of inflammation may be studied as to extent and degree by the roentgenologist. Syphilitic and tuberculous lesions of the larynx will generally be shown by roentgenologic study, but their differential diagnosis cannot be made by the roentgen ray alone. Roentgen study is especially helpful in cases where there is stenosis, compressive or otherwise.

5. Benign growths of the vocal cords will almost always show as rounded shadows projecting into the lumen of the ventricle of the larynx, the vestibule or the subglottic airway. It is chiefly in the larger growths that diagnostic roentgen study is of practical value; in such cases it is indispensable.

6. Carcinoma may be profitably studied throughout its course by the roentgen ray. As Coutard has shown, roentgenographic study is helpful in deciding upon the method of treatment, and it is also helpful in recording the *effect* of treatment.

7. One of the most important uses of roentgenography of the neck is in the determination of the proper position, size and shape of tracheotomy tubes and laryngostomy apparatus.

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LXXVII

FURTHER OBSERVATIONS ON THE USE OF SPECIFIC IMMUNE SERUMS IN THE TREATMENT OF STREPTOCOCCIC INFECTIONS*

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Two years ago it was my privilege to address this Association on the subject of specific treatment in streptococcic infections.[1] At that time I stated that the blood of certain patients convalescing from streptococcic infections seemed to contain immune substances which could be utilized in the treatment of clinically dissimilar infections of streptococcic etiology, and suggested a method of selection of high titer serums for therapeutic use in such instances. While human convalescent serum had previously been widely used in the treatment of identical infections, such as scarlet fever derived serum in the treatment of scarlet fever, its use in the treatment of various streptococcic infections had certainly not been general, if it had been used at all.

A careful search of the older literature reveals but two references applicable to the subject. One, by J. Boente,[2] mentions the use of convalescent scarlet fever serum to combat the toxemia of patients with streptococcic infections; the other, a brief report by H. Killian,[3] recounts the use of convalescent scarlet fever serum in a series of cases of streptococcic sepsis. These references are dated 1927 and 1928, respectively, and have been briefly referred to in a recent paper by Thalhimer and Levinson,[4] but should also be noted here in order to complete the record. In the paper last mentioned, published subsequent to my own, Thalhimer and Levinson recount their experience with convalescent scarlet fever serum in the treatment of diverse streptococcic infections, commenting on the procedure very favorably, but limiting their discussion to the use of pooled serum only, no method of serum selection having as yet been instituted by them. Champ Lyons, on the other hand, in a very recent paper,[5] goes into considerable detail in his description of a method of selection of donors for transfusion in hemolytic streptococcic infections, basing his con-

*Read at the meeting of the American Laryngological Association, Detroit, May 25, 1936.

clusions on the fact that some individuals possess, in their serum, the specific opsonizing antibody which is essential to the process of phagocytosis and intracellular digestion of streptococci. This fact he has most satisfactorily demonstrated, and his method of selection is essentially that of determining the opsonic index of the donor against the particular invading organism to be combated. It should be further mentioned that Lyons at the same time treats the toxemia, when necessary, by the use of commercial (horse serum) antitoxin, the selection of which is made by means of the Schultz-Charlton test of specific blanching.

My own work has been carried out along the lines laid down in my original paper, and is based upon one proven fact and one strong presumption. The fact which I now consider to have been proven by innumerable clinical demonstrations in my own hands and those of others is that following streptococcic infections human convalescent serum frequently contains available antibodies which may be used in the treatment of other streptococcic infections characterized by clinical manifestations essentially different from those from which the donor has recovered. The presumption to which I still cling with renewed and increasing faith is that the agglutination test is a most valuable means of selection of serums for use in the treatment of individual cases.

The work and conclusions of Lancefield,[6 7] Lancefield and Hare,[8] Swift, Lancefield and Goodner[9] and of Griffith[10] give much encouragement to this presumption, although uniformly favorable clinical experience is by far the most convincing of all arguments. The work of Lancefield and collaborators is based upon the former's discovery that "hemolytic streptococci can be differentiated serologically by means of the precipitin reaction into distinct and sharply defined groups." And that practically all such organisms associated with human infections fall into a single group, Lancefield's group A. For type differentiation in this group Lancefield "employs the precipitin test with acid extracts of the so-called M fraction—and suitably prepared immune rabbit sera." It may at first seem that the precipitin reactions of Lancefield can have little relation to agglutination tests as used clinically in my laboratory, but Lancefield herself observes that the results of type differentiation by her method parallel closely those reported by Griffith with his special slide-agglutination technic. Briefly, this procedure, as described by Griffith, consists in the production of immune bodies in experimental animals (rabbits) by means of repeated injections with suspensions prepared from cultures of various strains of streptococci, thus producing in the animals anti-

serums satisfactory for testing. When applied to suspensions of streptococci of unknown types positive agglutination reactions are taken to indicate type specificity and the organisms in question are accordingly so classified.

It is true that none of the authors referred to worked with the intention of selecting specific serums for therapeutic use, but for the purpose of classifying the organisms according to their serologic reactions and thus relating them experimentally to the epidemics and individual clinical manifestations with which they were known to have been associated. As a result of their studies and in conformity with the opinions of many laboratory workers and clinicians, both Lancefield and Griffith agree that the various streptococcic infections, although distinct clinical entities and covering a wide range of disease manifestations, may be found associated in different individuals with entirely different strains of the organism. For example, Griffith has shown that as many as thirteen distinct types of hemolytic streptococci, as differentiated serologically by slide-agglutination, were present in 222 strains cultured from a series of cases of scarlet fever. This may explain the fact that scarlet fever has proved to be the most prolific source of antistreptococcic serum in my work and that we have rarely failed to find among our scarlet fever derived serums at least one which was apparently specific in its therapeutic action against a given streptococcic infection. It doubtless also explains the fact, stressed in former papers on this subject, that every scarlet fever derived serum is apparently not as therapeutically potent, even against scarlet fever and its complications, as some may be.

Thus I think it must be accepted as proven that many different disease manifestations may result from invasion of the human body by streptococci of the same serologic type, and that quite similar clinical entities may be found associated with streptococci giving different type-specific reactions. And, therefore, since organisms causing identical disease manifestations vary widely in type-specificity, it is logical to conclude that the protective substances elaborated by different individuals suffering from the same clinical manifestations differ proportionately—not to mention the variation in immune reactions which would naturally be expected to result from different hereditary, constitutional and general physical conditions to be found in various individuals. This we have found clinically to be true, for there is great variation in agglutinating titer of serums from different individuals, and a proportionately great variation in therapeutic specificity when the serums are used in treatment of diverse streptococcic infections.

Griffith makes another observation which is both interesting and important in relation to the work under consideration. He has noted that his rabbit-derived serums invariably contain agglutinins which act upon heterologous members of the group. In other words, every serum contains agglutinins which are specific for organisms other than the one administered to produce them. The presence of these agglutinins he attributes to a group reaction in the inoculated animal, and he eliminates them from his type-specific antiserums by absorption with heterologous antigens—that is, with mixtures of other type strains of streptococci. These strains absorb the group agglutinins, leaving the serum type-specific for the homologous organism only.

This observation is significant, because it conforms with our own laboratory results and with my clinical experience. The serum of certain donors has been found to possess a high titer of agglutinins for almost any streptococcus, though not for all, and such serums are usually therapeutically potent in the treatment of almost any streptococcic infection. In such serums there is doubtless present a high content of group agglutinins, and since human beings respond more surely and more violently to streptococcic infection than rabbits, it is safe to assume that the group reaction represented by Griffith's experimental findings and by our own clinical and laboratory observations is more marked and more frequent in humans than in experimental animals.

Practically it is not of great importance to us as clinicians to determine type-specificity of organisms with which we are dealing. We are most interested in knowing whether or not the serum we wish to use will be highly specific therapeutically against the particular organism involved, but it is not essential to determine whether this therapeutic efficiency is a group reaction or a type-specific reaction. And incidentally, when the term "specific" or "specificity" is used in this work, it should be understood that unless otherwise indicated therapeutic specificity is meant, and not type-specificity as it relates to the particular organism in question. I mention this point because of the fact that in discussing the basic theories underlying this study with laboratory workers I have found that they tend to confuse my meaning relative to these terms, and to assume that it is type-specificity which I am endeavoring to determine. That is a problem for the research laboratory, and from my point of view is an academic one at the present time. The presence of agglutinins in a serum is certainly an evidence of the existence of protective substances in that serum, and I have no reason to think that group agglutinins

and associated antibodies are any less potent therapeutically than type-specific substances. My clinical experience certainly indicates that there is little of any difference—a conclusion which I believe to be amply substantiated by our laboratory studies.

It might be observed with reason that the agglutination test alone is an insufficient method of determination of therapeutic efficiency. This is undoubtedly true, for the test demonstrates but one protective action of the serum against the invading organism, and it is well known that bacteria have many deleterious effects upon the host and that these deleterious effects are produced by different agents, such as erythrogenic toxins, fibrolysins, hemolysins and leucocidins. It is probable, and to some extent it has already been demonstrated, that these different agents require different protective and defensive reactions to combat them, but this need not concern us too greatly therapeutically, for there is at present no specific agent available for the neutralization of each different toxin, nor is there likely to be for some time in the future. We are daily faced with the practical problem of treating individuals sick with streptococcic infections, we now know that we have an agent which contains some if not all of the specific therapeutic agents needed, and we have also a method of estimating its relative therapeutic efficiency. A serum possessing a maximum of agglutinins elaborated as a result of the response on the part of the donor to invasion by a certain microorganism can reasonably be assumed to possess every other property which the human economy would need to enable such a donor to have successfully combated the infection and recovered. In the present state of our knowledge this is only an assumption and certainly not provable. But it is reasonable, and, in fact, almost self-evident. It is, therefore, also reasonable to assume that such a donor's serum will contain the substances needed to combat another similar infection, whether the symptoms of that infection are due to the action of fibrolysins, hemolysins, leucocydins, erythrogenic toxins, or what not.

In attempting to check our agglutination tests with other indicators of therapeutic specificity, we have endeavored to demonstrate the presence of the opsonizing antibody in a series of cases. Here we have determined opsonic indices by means of a method worked out by my associate, Dr. McConnell, and described by her in a recent paper.[11] We have found that the presence of the opsonizing antibody closely parallels that of the agglutinin, and here again clinical results have quite faithfully borne out our laboratory findings.

Nothing that has been said must be construed as an argument against the use of pooled serum, but merely as an exposition of the

merits of selected serums and of the agglutination test as a method of selection. My own work was originally based upon the use of unselected serums, and my first paper upon this subject specifically mentioned that in the interim between the clinical diagnosis of a streptococcic infection and its positive laboratory identification the empirical use of any available convalescent streptococcic serum should be resorted to. In a subsequent paper[12] I discussed the use of pooled serum at some length, and in the past two years the Children's Hospital of Denver, to mention but a single example, has dispensed literally gallons of pooled convalescent serum for use in Denver and vicinity, and the results, both within and wtihout that institution, have been of unquestioned value. This serum has all been taken from scarlet fever donors, and frequent tests in my laboratory have revealed that its agglutinating power is never less than plus one nor more than plus two, on the basis of four. Its therapeutic action seems frequently to be almost as effective as that of selected serum and its use is, therefore, not to be condemned, but to be commended and encouraged.

On the other hand, the use of pooled serum is necessarily empirical, so far as specific therapeutic value is concerned, and if selected serum of high therapeutic titer is available we are not justified in stopping with the simpler and easier method. Streptococcic infections are frequently of such severity and are so difficult to treat successfully that we need to avail ourselves of the most accurate and scientific means at our disposal. For that reason, and because therapeutic application has convinced me that specificity is in considerable degree indicated by agglutination, I continue to recommend to clinicians the use of agglutination tests as indicators of therapeutic specificity. While I use selected high-titer serum and pooled serums without agglutination when necessary, I invariably prepare to administer the more effective selected serum if it proves to be needed.

A brief description of methods used and results obtained in this work may be permissible here, although I would refer those vitally interested to former papers for details. Our serums are taken from any and all individuals recently convalescent from streptococcic infections of any type, scarlet fever donors being by far the most prolific source of supply. Blood in amounts from 200 cc. to 500 cc. is taken, the serum separated, and serologic tests for syphilis are made. It is then Seitz filtered, cultured and a preservative in the form of 0.3 per cent cresol U. S. P. added. It is bottled, cultured a second time, and stored in the refrigerator. At the same time, a small sample is set aside for agglutination purposes. It might be added that the

agglutinating titer of each donor's serum is taken before adopting said donor for use and if his serum proves to be disappointing in its serologic reactions he is dismissed.

Upon encountering a patient suffering from what appears to be a streptococcic infection, an immediate dose of high titer selected serum or of pooled serum is administered empirically, and a culture of the lesion taken. After twenty-four hours, if the culture proves positive for streptococci, agglutination tests are carried out and a serum of high agglutinating titer selected for further use. A sufficient number of different known high titer serums are kept on hand so that we seldom fail to find one apparently satisfactory, and if further treatment is necessary this selected serum is administered. Total dosage varies from 20 cc. to 300 cc., and in desperate cases as much as 100 cc. has been given in the first twenty-four hours. Ordinarily such large dosage is unnecessary, and I am in some doubt as to whether total dosage in excess of 150 or 200 cc. is of much added value. While I frequently give larger amounts where it appears to be necessary, I feel that in the last analysis the patient must himself overcome his infection, doing the final housecleaning with his own immune processes, and that no amount of artificially administered passive immunity will take the place of an absent or nonexistent active immunity. The serum seems simply to augment but not supplant the patient's own immune processes, apparently acting as an antitoxin in addition to promoting the defensive mechanism of the body against the organisms themselves. It should be mentioned that immune transfusion may be used as a supplement to serum administration, or as an entire substitute for it. Here the same principles apply, and the choice is dependent upon the factors of availability and the patient's need for blood elements in addition to immune substances.

In advocating a method of specific treatment which may be spectacular in its results, and which is on the whole simple and easy of application, I feel that it is important to call attention to certain limitations and issue certain warnings. The outstanding failures in treatment by this method in my hands have been the cases of streptococcic meningitis. While most of my series of seven cases have been late and hopeless, a few have offered some hope of cure with specific therapy. All have been most carefully treated by both intravenous and intraspinal injection of large doses of selected serum as an adjunct to adequate surgical drainage of local suppurative areas. In every instance the treatment has proved to be a conspicuous failure, not only in its final result but by complete absence of even temporary

symptomatic improvement. Several cases of overwhelming toxemia, notably complicating scarlet fever, have made no response whatever to the serum, going on to fatal termination without the slightest improvement. In three fatal cases of pancarditis with positive blood findings serum administration seemed to result in slight symptomatic improvement which was invariably lost within forty-eight hours. Other cases of general sepsis have been marked by one consistent observation—an observation which I have emphasized before and will repeat. The serum will not and must not be expected to eliminate active foci of infection. For example, a septic thrombus must be removed from the lateral sinus and the sinus drained before improvement can be expected, and the administration of serum in the fatuous hope of avoiding surgery in such cases is only to be condemned.

A recent case of less seriousness but of extraordinary personal interest to me illustrates these points very clearly. A physician suffered an attack of streptococcic tonsillo-adenitis of more than usual severity and was given early and adequate dosage of serum to combat the infection. Results were so satisfactory that the temperature was down in twenty-four hours, the symptoms clearing rapidly under further dosage of selected serum. An acute nephritis supervened, however, which did not improve, and cultures from the tonsils were consistently positive for hemolytic streptococci in spite of complete clearing of all local symptoms. After unavailing administration of additional serum, removal of the tonsils resulted in clearing of the urinary findings in a period of two weeks, and they have since remained clear. Such instances could be multiplied indefinitely, but these are sufficient to illustrate the fact that drainage or removal of both original and secondary foci should be resorted to under the same rules of surgical procedure that would apply without serum therapy. Serum may be used as an adjunct in surgical cases if necessary, but should never be depended on as a substitute for needed surgery, or when surgical indications supervene.

It is much more pleasant to recount one's successes than one's failures, and I am tempted to report some of the more outstanding instances of successful serum therapy. A long list of these could be appended, but the method has been so thoroughly tried out at the hands of others and so unanimously adopted where such trial has been undertaken that it seems to me a needless waste of time to do so. It is sufficient to summarize the results of successful treatment.

Slight if any improvement is observed in the first twelve hours after administration. During the second twelve hours the merest

hint of improvement may be noted, though this is often debatable. In the third twelve hours the progress toward cure is frequently phenomenal, if the serum is to prove effective. If sufficient dosage has been administered, and no improvement is noted in the third twelve-hour period I am inclined to think that treatment by this means will be unavailing. At the end of forty-eight hours, if treatment is to accomplish anything, improvement is invariably self-evident and further progress as a rule rapid and uneventful, with much shortened convalescence.

The method has been successfully used in a wide series of cases, ranging from streptococcic throat infections and their complications to streptococcic peritonitis, but always in conjunction with needed surgery where suppurative lesions have been present, or even in the presence of active foci. In over 1,500 administrations of serum at my hands there has been no single instance of even an unpleasant reaction, not to mention a serious one. The worst thing to be said of it is that it is somewhat troublesome, but I hope that those of you who have not tried its use and who consider the selection of serums a procedure of too great difficulty, will at least arrange to have a small serum pool established in your community. Simply collect serum from at least five recently recovered scarlet fever patients, preferably those who have had reasonably severe attacks, pool this serum, prepare it as already described, and use the pooled serum in a small series of streptococcic infections. If the therapeutic results are disappointing, discard the method, but if they are encouraging, proceed the short step further required to set up the necessary machinery for routine agglutination tests, persuade your laboratory heads to institute routine agglutinations and either confirm or condemn the conclusions here offered.

There is much to be learned about the streptococcus and about streptococcic infections, as we are still in a state of abysmal ignorance on the highly complicated subject of specific immunity and resistance to infection. These matters will require infinite labor for their elucidation, but in the meantime, if my importunities persuade you to adopt the simple expedient of using pooled convalescent scarlet fever serum in the treatment of your cases of streptococcic infection, and if your successes with this procedure encourage you to the application of the routine I further recommend, I am sure that your results will be more than gratifying, and I will derive the high satisfaction which comes from the knowledge that one's labors have not been in vain.

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LXXVIII

VERTIGO IN BRAIN TUMORS, WITH SPECIAL REFERENCE TO THE RESULTS OF LABYRINTH EXAMINATION*

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Vertigo in brain tumors is commonly considered as a general symptom of increased intracranial pressure, and its genesis is explained as due to the action of the pressure upon the labyrinth and its rhombencephalic centers. It was, however, already pointed out by Leidler[1] that the mechanism of vertigo in diseases of the cerebrum may be much more complex. The aforementioned view neglects the possibility that the vertigo may be caused, at least in some cases, by injury to a cortical center of the labyrinth. If one bears in mind that stimulation of the labyrinth gives rise, as does stimulation of other sense organs, to conscious sensations, such as the perception of position and movements, one should expect that the labyrinth too has a cortical representation, and that stimulation of such a focus may cause vertigo. Unfortunately, our knowledge of the anatomic pathways connecting the rhombencephalic vestibular nuclei with the cortex is quite hypothetical, and the few clinical experiences pointing to the existence of a cortical center of the sense of equilibrium are quite contradictory. While the experiences of Hitzig-Wollenberg,[2] and Bruns[3] suggest the importance of the frontal lobe and of the region of the central gyri respectively for the perception of labyrinthine impulses, it may be inferred from the statements of Bechterew,[4] M. H. Fischer[5] and Foerster[6] that the parietal lobe has to do with such perceptions; a third group of observations (Dana,[7] Mills,[8] Meyers,[9] and Knapp[10]) indicate that the temporal lobe may be the site of a labyrinth center. In view of this uncertainty of our present anatomic and clinical knowledge one of us (Spiegel[11]) tried experimentally to study the entrance of labyrinthine impulses into the cerebral cortex. The excitability of the various sensory cortical areas was increased by

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localized strychninization in cats and dogs, and after such preparation the animals were rotated on a Bárány chair. After strychninization of the parts of the temporal lobe (posterior supra- and ectosylvian gyrus) stimulation of the labyrinth by rotation induced epileptiform convulsions, while strychninization of the parietal or frontal lobe did not have such an effect. It was concluded that a cortical center of the labyrinth must be sought in parts of the temporal lobe. If this region becomes hyperexcitable, e. g., by localized strychninization, the labyrinthine impulses entering this region may radiate to neighboring motor centers so that epileptiform convulsions result. These experiments were confirmed by L. Aronson.[12] It is of interest to note that, independent of these experiments, Stephan Poljak[13] (1932) traced corticopetal fibers that end in the posterior part of the Sylvian fissure, behind the projection field of the cochlea in monkeys, after having produced subcortical lesions. He ventures the possibility that this system may have something to do with the sense of equilibrium, but he does this without studying this question by experiments. In further experiments of Spiegel the cortical action potentials (the electroencephalogram) were studied after stimulation of the labyrinth. A postrotatory increase of the cortical potentials was found. In the parietal lobe this increase depended upon the intactness of the temporal and of the frontal lobe and was absent after combined extirpation of these lobes. In the frontal lobe this postrotatory increase of potentials was still found after bilateral extirpation of the temporal lobe, but a definite effect of the rotation failed to appear, if this extirpation was combined with high transverse section of the spinal cord. If the potentials were led off from the temporal lobe, rotation was followed by their increase, also after vagotomy, severance of the spinal cord, extirpation of the parietal or of the frontal lobe. These experiments seemed to indicate that the labyrinth is represented in the cortex, particularly in parts of the temporal lobe, but that a part may also reach the frontal lobe by way of the rubrothalamo-frontal tracts. In these latter systems an interaction with impulses originating in the spinal cord may occur.

The question presents itself as to how far these results of animal experiments may be applied to the human brain. In fact, these experiments were followed by a number of clinical observations indicating that also in man labyrinthine impulses may enter the temporal lobe. Barré, Vincent and Helle[14] report a case of temporal lobe tumor with typical turning sensation. Penfield and Gage[15] described tumors of this region that induced epileptiform convulsions with attacks of dizziness. Such attacks could also be produced by electrical

excitation of the affected region of the cerebral cortex. The study of certain cases of so-called genuine epilepsy may also shed some light upon our problem. Stauder,[16] for instance, observed such cases in Bumke's clinic. In one patient the aura was characterized by a quick succession of acoustic and olfactory hallucinations, somewhat later a severe "vestibular syndrome" developed (everything turned around the patient). Finally the optic perceptions were also affected; simultaneously amnesic-aphasic and paraphasic disturbances respectively appeared. In a second patient there was during the aura a succession of olfactory, vestibular and paraphasic disturbances. The author interprets his observations as confirming the assumption of a cortical representation of the vestibular system in the temporal lobe, in agreement with Spiegel's experiments on animals.

These are, however, only single observations. A systematic study of the cortical representation of the labyrinth in the human brain seemed, therefore, desirable. In chosing the material one has to bear in mind that, according to Aronson's experiments, each labyrinth has a bilateral cortical representation, as has the cochlea. In unilateral destruction of the cortical labyrinth center, one can, therefore, hardly expect to find deficiency symptoms, or one may find such symptoms only if particular methods are applied. Unilateral cortical diseases may easier elicit distinct clinical symptoms, if such diseases have a stimulating effect upon the cortical centers. One may therefore hope to get some information about our problem by studying how far vertigo in brain tumors depends on the localization of the tumor. From the tumor material of the First Surgical Clinic of the University of Vienna* (Prof. v. Eiselsberg, Prof. Ranzi), we selected only those cases where the localization of the tumor was established by operation or postmortem examination and in which a single more or less circumscribed process was found. The material is, therefore, limited to 192 cases; 41 of these belong to the frontal lobe (with exception of the central gyri), 52 to the central gyri, 28 to the parietal lobe, 46 to the temporal lobe, 11 to the occipital lobe, 14 to the basal ganglia and the corpus callosum.

In the analysis of the material, two points seem of importance. First one has to exclude as much as possible the distant effects of pressure upon the labyrinth, the cranial nerves and the brain stem. Second one has to decide which cases should be designated as vertigo positive. The differences in the literature concerning the frequency of vertigo in tumors of a certain part of the cerebral hemispheres

*We wish to express our sincere thanks to Prof. Ranzi for permission to study the material of the clinic.

(compare, for instance, in regard to the temporal lobe, the figures of Artom[17] and Marburg[18]) are for the most part due to the fact that some authors consider only those cases as vertigo positive that have a true turning sensation, while others include also types of unsystematized vertigo. One has to bear in mind that the labyrinth is a receptor not only for sensations due to rotation but also for those due to rectilinear (progression) movements and for sensations due to various positions of the head in space. In fact, patients with diseases of the labyrinth and still more those with lesions of the rhombencephalic nuclei and of pathways of this organ, complain not only about turning sensations but also about sensations of rectilinear movements in horizontal or vertical direction; the patient has, for instance, the sensation of plunging into an abyss, or of being lifted or pushed or pulled to the left or right side. In other cases one finds delusions regarding the position of the head; the patient feels as though the head is in an oblique position. Finally, the sensations may be confused, and lack any direction (Abels,[19] Brunner,[20] H. M. Fischer[21] and Stein.[22]) It seems, therefore, necessary to consider as vertigo positive not only cases that have a typical turning sensation, but also all cases with types of vertigo as just described.

Table 1 shows the material before an analysis was attempted. The values were corrected in the following way. If a tumor was, for instance, originally classified as parietal lobe tumor but also belonged partly to the occipital lobe, .5 was deducted from the number of parietal lobe tumors and .5 was added to the number of occipital lobe tumors. This procedure was repeated in the column "cases with vertigo," if the particular case had vertigo. If the tumors belonged to three lobes instead of one, two-thirds was deducted from the one lobe and one-third was added to each of the other two lobes.

TABLE I

Region	Total No. of cases	Cases with vertigo	Distribution upon the lobes corrected: Total No. of cases	Distribution upon the lobes corrected: Cases with vertigo
Frontal lobe (without central gyri)	41	6(14.2%)	42	7(16.6%)
Central gyri	52	11(21.1%)	50.5	11(21.7%)
Parietal lobe (without central gyri)	28	8(28.5%)	28.3	7.7(27.1%)
Temporal lobe	46	34(73.7%)	43.1	30.2(70.0%)
Occipital lobe	11	3(27.2%)	14.1	6.1(42.8%)
Basal ganglia and corpus callosum	14	9(64.2%)	14	9(64.2%)
Total	192	71(36.9%)	19.2	71

A comparison of the figures given in the last column of Table I shows that the complaint of vertigo was most frequent in tumors of the temporal lobe. The figures of this table, however, do not give as yet a correct conception of the rôle of the cortical localization of the tumor in the genesis of vertigo, because a large part of these cases was complicated by pressure upon the labyrinth.

TABLE II

CASES WITH EXAMINATION OF THE LABYRINTH

	Number of cases	Overexcitability of the labyrinth without vertigo	Vertigo with abnormal excitability of the labyrinth (over-excitability. Differences in excitability between both sides)	Vertigo with normal excitability of the labyrinth and with cerebellar symptoms, nystagmus, etc.	Vertigo with normal excitability of the labyrinth and without cerebellar symptoms, nystagmus, etc.	
Frontal lobe (without central gyri)	18	4	2	2	2	10.2%
	corrected: 19.5	corrected 4.2	corrected: 3			
Central gyri	18	4	1	4	3	17.6%
	corrected: 17.0					
Parietal lobe (without central gyri)	12	1	2	2	2	
	corrected: 13.7	corrected: 1.3	corrected: 2.5	corrected: 1.9	corrected: 1.4	10.2%
Temporal lobe	31	4	13	12	7	
	corrected: 25.7	corrected: 3.5	corrected: 10	corrected: 10.8	corrected: 6.8	26.4%
Occipital lobe	7	2	1	1	1	
	corrected: 10.1		corrected: 2.5	corrected: 2.3	corrected: 1.8	17.8%
Basal ganglia	9	1	4	1	1	11.1%

It seems, therefore, necessary to devote special consideration to those cases in which the labyrinth function was tested.* Table II shows in the first column that in all groups of tumors cases are found that had no vertigo, although an increased excitability of the labyrinth existed. For an analysis of our problem the next column is of greater importance, showing that the appearance of vertigo was explainable in a large part of this material by abnormal excita-

*The results of the ear examination until 1921 were published in detail by J. Fischer, Monatschr. f. Ohrenheilk., 55:371, 1921.

bility of the labyrinth (mostly overexcitability, in some cases differences in excitability between both sides). Yet there still remain cases of vertigo with normal result of the labyrinth examination (last columns). Sometimes such an examination may fail to detect slight differences in the state of excitation of the labyrinth, but such differences may induce vertigo and become manifested by the appearance of spontaneous nystagmus. Such cases, as well as cases with diplopia, must, of course, also be excluded. One has also to bear in mind that patients with cerebral tumor often complain of vertigo, if there exists a disturbance of equilibrium or a tendency to fall due to disorder of the cerebellar function that is caused by pressure upon this organ or upon its branches, or caused by injury to the long descending cerebral pathways. Thus in Table II all cases were finally excluded in which spontaneous nystagmus, diplopia or disturbances in equilibrium indicating a cerebellar disturbance might have been responsible for the appearance of vertigo. The last column contains these cases in which the vertigo could not be explained, either by the result of the labyrinth examination or by the other neurologic symptoms. In calculating the percentage, the figures were again corrected in that .5 (.3) was assigned to each of the lobes concerned if the tumor was localized in two or three lobes. Also if the aforementioned criteria were applied in the analysis of the material it resulted that tumors of the temporal lobe were most frequently associated with vertigo (last column, Table II.*)

It was further tried to ascertain as to whether within the various lobes some localizations were more conducive to the appearance of vertigo than others. In this respect only this can be said. As to the central region, vertigo occurred more frequently in tumors of the ventral parts than in growths of the dorsal parts. In eleven tumors close to the dorsal margin, vertigo was only twice noticed, while it appeared four times in twelve tumors of the ventral parts of the central region. The reverse seems to be valid in temporal lobe tumors, in so far as the smallness of the material permits such conclusions. In twenty cases a dorsal and a ventral localization respectively could be distinguished. The group of dorsal tumors[13] showed vertigo ten times (77 per cent), the group of ventral tumors[7] showed this symptom four times (57 per cent). The material did not permit to ascertain as to whether growths of cranial or of caudal parts of the temporal lobe more frequently induced vertigo.

*The percentages of the column show the frequency of vertigo in each single group of tumors and not in relation to the total number of cases. One may not expect, therefore, that the sum of the percentage figures in the last column is 100.

It is only with the greatest caution and reserve that one may draw conclusions from these observations in respect to the cortical localization of labyrinthine centers. Such a caution is dictated by the relative smallness of the material and by the uncertainty to which a localization is subject that is based upon diseases complicated by increased intracranial pressure. At any rate, one can not neglect the fact that there exists a different frequency of vertigo in tumors of the various areas of the cerebral cortex. If this symptom were simply due to a distant effect (pressure) upon basal nerves, labyrinth or the cerebellum, one should expect that more ventrally located tumors would be more frequently associated with vertigo than tumors close to the cortex. This was found in tumors of the central region, but just the opposite in tumors of the temporal lobe. The striking frequency of vertigo in tumors of this region found even after cases with vestibular overexcitability, nystagmus, diplopia or pseudocerebellar ataxia were excluded, seems to indicate that the temporal lobe has something to do with the perception of labyrinthine impulses, in the human brain, as well as experimental animals. Yet this should not be interpreted as meaning that the temporal lobe is the only region where vestibular impulses enter. It was already mentioned that the participation of the frontal lobe in these perceptions must also be considered. The anatomic studies of Monakow[23] indicate that the cortical projection of the nucleus ruber must be sought in the frontal lobe and the centro-opercular region. Although the doctrine of Rademaker,[24] that the nucleus ruber is the center of the labyrinthine righting reflexes was recently invalidated (Mussen,[25] Ranson and Ingram,[26] and Keller and Hare[27]), at least that much is certain that the nucleus ruber receives labyrinthine impulses, mixed with impulses of spinal origin, by way of the superior cerebellar peduncle. Moreover, L. Alexander and J. G. Whitaker[28] described direct tracts from the vestibular nuclei to the nucleus ruber in dogs in which one of us (Spiegel) had injured the vestibular nuclei. Thus one may assume that labyrinthine impulses, mixed with impulses of spinal origin, may reach the cortical representation of the nucleus ruber in the frontal lobe and centro-opercular region respectively. This may explain the relatively high frequency of vertigo in tumors of ventral parts of the central region.

SUMMARY

The observations on brain tumors seem to corroborate, also with regard to the human brain, the assumption of a representation of the labyrinth in the cerebral cortex, particularly in the temporal lobe. Parts of the frontal lobe, especially the centro-opercular region, must

also be taken into consideration as a place where vestibular and spinal impulses, joined in the subcortex (cerebellum-ruber system) may enter. The conception that vertigo in brain tumors is only a general symptom of increased intracranial pressure seems to need a revision. In a large number of cases symptoms of choked labyrinth, hyperexcitability of this organ, differences in excitability between both sides, nystagmus, diplopia, and cerebellar disturbances are found, and the appearance of vertigo is explainable as due to these effects of increased intracranial pressure. Yet there remains a group of cases where such an effect of pressure upon the labyrinth or upon the brain stem is absent. In these cases, at least, it seems not unreasonable to assume that the vertigo may appear as a local symptom of the cerebral cortex due to direct lesion (stimulation) of the aforementioned cerebral areas, or due to pressure upon these foci by tumors in neighboring regions. In general, it seems that tumors close to the Sylvian fissure induce vertigo more easily than do tumors more distant from this fissure.

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LXXIX

OTOMYCOSIS: AN INVESTIGATION OF EFFECTIVE FUNGICIDAL AGENTS IN TREATMENT*

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TUSCALOOSA

The fact that many types of molds commonly found leading a nonparasitic existence in air, soil and water, and upon animal and vegetable material, frequently invade the human body and produce definite pathology has been established. Such conditions as ulcerations, massive growths (mycetomas), infections of the beard, the nail bed, joints, infections of the tongue and of the respiratory tract have been described. The relationship of all of these as primary invaders has not been well established, since animal inoculations that have been tried with the isolated fungi have almost invariably yielded negative results. However, many cases of mold infections of the respiratory tract of man, cattle, swine and birds are recorded with clear cut clinical symptoms. Culture of exudates; of gross lesions where necropsy was performed, and the study of the histopathology of such has left little doubt as to the etiology involved.

In the present paper, interest is centered upon the rôle these seemingly harmless saprophytes play in producing lesions of the external auditory meatus and commonly known as otomycosis. Of the molds encountered, aspergilli are most common. That otomycosis occurs more frequently than ordinarily suspected and is often unrecognized even by physicians, and that it may cause considerable discomfort, is well illustrated in the case of a colleague who was one among ten cases studied and reported upon by the authors at the annual meeting of the Alabama State Medical Association in 1929. Quoting from the case reports:

"Dr. W. was treated at intervals for several years for an eczematous condition of the external canals. They would fill with a

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moist, dirty, membranous material and become sore. He would clean them as best he could or get some convenient colleague to do it for him. Microscopic and cultural studies from this and other cases studied showed that material taken from the ear was a mass of mold growth; in all cases identified as aspergilli, and which under adequate treatment cleared up without further trouble."

Methods of treatment and the investigation concerning those which appear most adequate will be subsequently considered.

Reviewing the literature, we find that much has been written upon the incidence of otomycosis and the factors involved as well as methods of treatment.

Wreden[2] mentions that Mayer, 1844; Pacini, 1864, and Schwartz, 1865, were first to study the condition. Wreden observed it occurring among Russians of Petrograd. He noticed that those most affected were the poorer, undernourished class who lived in poorly ventilated, damp houses, dampness apparently being favorable to the growth of the mold. While this inference is undoubtedly true, subsequent studies show that otomycosis is not confined chiefly to those individuals less favored economically.

Bar,[3] in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, states that the incidence is relatively small, being 0.1 per cent of the population. Bezold[4] puts it at 1.5 per cent. It is likely that these figures fail to approach the actual number of cases existing, owing to failure to recognize such and because of confusion with other forms of otitis.

Since the recognition of the first case, reports of its occurrence have come from many corners of the world.

Symptoms of otomycosis are variable. There may be only a sense of fullness, or itching, burning, slight pain, tinnitus and impairment of hearing. Whether symptoms are mild or exaggerated, the fact remains that individuals so affected are in need of some form of treatment that will relieve discomfort and effect a cure. A great variety of aspergilli have been isolated and studied from such cases. It would seem that the question of determining the species of aspergilli present, whether single or mixed species, would be of academic interest only, unless some species are more resistant to treatment than others. If detecting the mere persence of aspergilli is the only question involved, then simple microscopic examination without staining will suffice. If, on the other hand, certain species are more resistant than others to treatment, which consists essentially in thorough clean-

ing followed by the application of some efficient fungicidal agent offering maximum effect with minimum discomfort and tissue irritation, then the question of treatment becomes more involved. Many of these cases are complicated by accompanying invasion of pyogenic cocci, particularly staphylococcus aureus. In such cases combined fungicidal and bactericidal action is essential. This question is considered, in the present investigation, by determining the action of various chemical agents upon a variety of species and upon clinical cases when found efficient in their action.

Treatment of otomycosis from the first has consisted chiefly in mechanical removal with fine forceps or an applicator followed by thorough irrigation. Secondly, destruction of the offending invader, and thirdly, allaying the inflammation which their growth has caused. If we can find an efficient agent fulfilling the second requirement without tissue damage, then Nature will do much in the third instance towards a successful and satisfactory culmination.

Of the many fungicidal and bactericidal agents used, varying degrees of success have been obtained; that is, their use has allayed and not cleared up the condition; they have required multiple applications, or they have acted as tissue irritants; they have been ideal in the hands of one worker and not with others.

Upon reviewing the literature dealing with otomycosis from 1844, when first described by Mayer,[2] we find mention of some sixty different chemicals, used since that time, either alone or in combination, in an effort to give relief or effect a cure. Time nor space will not permit consideration of each. It is interesting to note, however, that Schwartz,[5] 1867, recommended cleansing with warm water followed by applying two leeches inside the tragus. This was subsequently followed by an aqueous solution of lead acetate. The whole took six weeks to effect a cure.

Alcohol in varying strengths has found wide application. Burnett,[6] as early as 1874, recommended three installations of an aqueous solution of nitrate of silver, 100 grams to the ounce, followed by 90 per cent alcohol to be used daily for one month. He states that the continued use of alcohol or of any other parasiticide is indicated in any case of growth of aspergilli in the ear.

As recent as 1930 Cavadas[7] stated, "Luckily alcohol, which is used so commonly in chronic otitis media, is a specific for fungus affections and in this way many unrecognized cases of aspergillosis are cured."

Other substances mentioned are:

Powdered boric acid, borax or boric acid in combination with chinoline salicylate.

Lavage of the canal several times daily with an aqueous solution of sodium bicarbonate or borate with bichloride.

Insertion into the meatus of wicks of sterile gauze saturated with alkaline solutions.

Internal use of alkalis:

Alcohol boric acid solution or alcohol bichloride.

Hydrogen peroxide-alcohol mixture:

Mercurochrome, also a solution of methylene blue or carbol fuchsin with the addition of resorcinol.

Alcohol-salicylic acid solution, silver nitrate in 90 per cent alcohol, and a weak aqueous solution of zinc sulphate.

Miller,[8] writing in the Journal of the American Medical Association, April 2, 1927, reports the use of peroxide alone and then 50 per cent alcohol. The former was tried for three days, the latter for one month with no results. The patient had visited many clinics. Two per cent salicylic in 95 per cent alcohol finally cleared up the condition.

Other forms of treatment consist in the use of weak carbolic solution, iodine solution, oil of cade, and potassium iodide internally. Of all the combinations used, 2 per cent salicylic in 95 per cent alcohol has found most wide application.

In 1929 the authors[1] reported the successful use of 2 per cent thymol in 95 per cent alcohol in ten identified cases of otomycosis, the use of which was suggested through an article by Dr. Harold B. Myers[9] in the Journal of the American Medical Association, November 26, 1927, wherein he reported marked fungicidal action of thymol and the volatile oils of cinnamon and clove upon yeast cultures from lesions on the hands of fruit gatherers. He also reported equally good results in the use of thymol upon cultures of actinomyces hominis.

Further, Dr. Searcy,[1] the same year, successfully treated a case of actinomyces of the jaw and later two others by injecting 10 per cent thymol in olive oil into fistulous tracts extending to the bone, packing with gauze saturated with the same solution. Fifteen grains of thymol by mouth at bedtime with no evening meal was also given. Since the initial use of thymol in alcohol, successful treatment of

many cases of otomycosis with this has been instituted by Dr. Searcy and its value clinically proven. The use of thymol in alcohol and of other suggested fungicidal agents, either alone or in combination in a search for the ideal, has suggested a comparison of these upon cultures of various species of aspergilli similar to those we have isolated from cases of otomycosis, and to test in vivo the clinical value of such as have proven satisfactory in vitro; further, to determine the minimum strength of such germicidal agents yielding maximum results without discomfort in actual cases.

In order to determine the relative fungicidal action, the agar cup plate method of determining the value of antiseptics, as outlined by Allen and Wright,[10] was decided upon.

Briefly the method consists in seeding melted standard beef extract agar in flasks, after it has been cooled to 45 deg. C., with suitable suspensions of the test organisms and the subsequent addition of 10 per cent sterile beef serum. Following thorough mixture, thirty cubic centimeters were poured into sterile petri dishes and allowed to harden, after which a disk 2 cm. in diameter was cut out of the center with a metal cork borer, sterilized by dipping in alcohol and flaming, leaving a perfect well or depression. The depression was sealed on the bottom with three to four drops of melted agar. Into this cup 0.5 cc. of the substance under investigation was placed. If a solid, the cup was filled level with the surface. Plates seeded with molds were incubated at room temperature for ninety-six hours; those with bacteria, forty-eight hours before readings were taken. The results are tabulated in the distances from the periphery of the cup, showing no growth. Measuring these distances in millimeters by means of dividers, and comparing to that produced by a 5 per cent phenol accurately titrated by the method of Koppescaar,[11] has been designated by us as the "Phenol Ratio," or "P. R.," which gives a whole number for expressing relative diffusibility and germicidal action.

For a time some difficulty was experienced in getting maximum mold growth on plates and in breaking up clumps for making dilutions. It was found, on trial platings, that 1 cc. of a 1:20,000 dilution for each 250 cc. of medium, as recommended by Allen and Wright for bacterial studies, was too high but that a like amount of a 1:5 dilution of the mold grown ninety-six hours at room temperature, in 50 cc. of suitable liquid medium containing approximately fifty glass beads, three to four millimeters in diameter, when well shaken, gave excellent results. Beads and cultural solution

were placed in salt mouth glass-stoppered bottles sterilized with gauze covered cotton plugs. Stoppers were sterilized in glass jars and were substituted for plugs when shaking cultures for dilution.

In the bacterial studies the method of Allen and Wright was adhered to. A twenty-four hour growth of S. aureus was used, and platings were made in nutrient beef extract agar pH 7.4.

In preliminary studies upon molds nutrient agar was used, then Sabourad's medium and finally Czapeck's Solution Agar and Broth, as given in that excellent treatise, "The Aspergilli," by Thom and Church,[12] published by Williams Wilkins, were adopted as best for maximum growth. The medium shown below is a synthetic one containing inorganic salts and sucrose but no peptone.

FORMULA FOR CZAPECK'S SOLUTION AGAR FOR GROWING MOLDS

Water	1,000 c.c.
Sodium nitrate	3 gms.
Potassium phosphate (K_2HPO_4)	1 gm.
Potassium chloride	0.5 gm.
Ferrous sulphate	0.01 gm.
Magnesium sulphate	0.5 gm.
Sucrose	30.00 gms.
Agar-agar	15.00 gms.

Sterilized in the autoclave at 15 lbs. for fifteen minutes.

With the concentration used in seeding mold cultures, growths were always entire instead of in individual colonies. (Fig. 1.) This concentration was purposeful, as it gave a more rigid standard than if higher dilutions had been used. When application of fungicides is made in the external auditory canal the mold growth is usually massive, and where such procedure as followed in the platings is used it seems to us to more nearly approach natural requirements for fungicidal action.

In addition to chemicals, mentioned in the literature, we have added others, such as cresatin, mentioned by Gill,[13] which has yielded good clinical results in his hands, naming it first in order of effectiveness but advising against its use in patients showing local sensitiveness to phenol. We have likewise found it very effective clinically, especially when mixed with thymol and alcohol. For proportions see Table 1.

Because of their well recognized bactericidal action merthiolate and metaphen have been used by us in combinations of thymol and alcohol and have proven excellent in cases complicated by bacterial invasion. See Table 1.

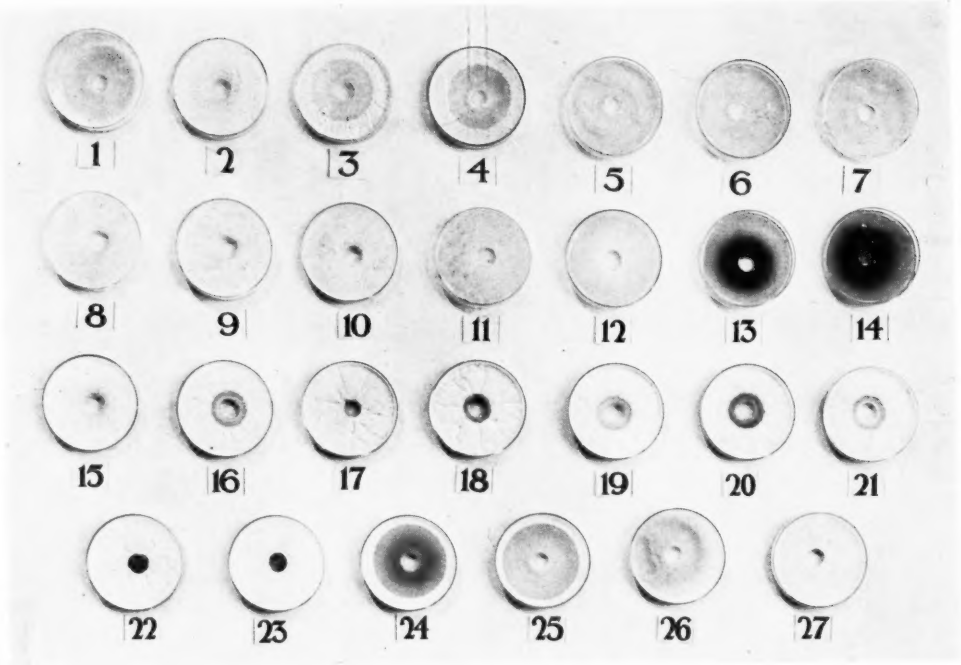

Fig. 1. Showing cup-plate method of testing fungicidal properties on aspergilli.

TABLE I

ACTION OF VARIOUS SUBSTANCES UPON SOME ASPERGILLI FOUND IN OTOMYCOSIS

ORGANISM Substance Employed	Phenol Ratio *N	 *W	 *G	 *F	
1. Alcohol absolute	0.0	0.0	0.0	0.0	Ethyl alcohol.
2. Alcohol, 95%	0.0	0.0	0 0	0.0	Ethyl alcohol.
3. Alcohol, 70%	0.0	0.0	0.0	0.0	Ethyl alcohol.
4. Alcohol, 50%	0.0	0.0	0.0	0.0	Ethyl alcohol.
5. Alcohol, 40%	0.0	0.0	0.0	0.0	Ethyl alcohol.
6. Alcohol, 30%	0.0	0.0	0.0	0.0	Ethyl alcohol.
7. Thymol, 2% aq. emulsion	1.6	2.5	1.9	2.2	Ethyl alcohol.
8. Thymol-alcohol, 95%	2.1	3.4	2.3	2.7	2% thymol in 95% alcohol.
9. Thymol-alcohol, 70%	1.6	2.5	1.9	2.2	2% thymol in 70% alcohol.
10. Thymol-alcohol, 50%	1.5	2.5	2.0	1.8	2% thymol in 50% alcohol.
11. Cresatin	2.6	3.4	2.2	2.0	Acetic acid ester of metacresol.
12. Thymol-cresatin-alcohol, 95%	2.6	3.4	2.2	2.1	Thymol gr. V. cresatin-alcohol, eq. pts. of each, dr. 2.
13. Merthiolate soln., 45%	2.0	2.7	1.7	1.4	Sodium-ethyl-mercuri-thio
14. Merthiolate tinct., 99%	2 0	2.6	1.7	1.6	Salicylic acid, 1:1000 dil.
15. Thymol-merthiolate, 95% alcoh.	2.1	2.9	2.3	2.7	2% thymol in merthiolate, 45—1:1000 dil. & alcohol eq. pts.
16. Castellani's solution	2.0	3.4	2.0	0.8	Basic fuchsin, gms. 1, boric, sat. aq. c.c.l. alcohol (95%), c.c.X., 5% aq. Phenol c.c. 90, resorcin gms. X.
17. Castellani's-thymol.	2.0	3.4	2.3	2.0	2% thymol in Castellani's
18. Gasoline	0.0	0.0	0.0	0.0	Gasoline. Low grade.
19. Gasoline-thymol	1.6	1.9	2.3	2.0	2% thymol in gasoline.
20. Sodsol	0.0	1.8	1.0	0.8	Naphtha cleaning solution.
21. Sodsol-thymol	1.8	1 6	1.3	1.5	2% thymol in sodsol.
22. Tincture iodine	1.5	2.4	1.7	1.8	
23. Thymol-boric-iodine	1.5	1.5	1.2	1.4	Thymol 2%, iodine 2%, boric 96%.
24. Carbolic-olive oil	1.8	2.0	1.5	1.9	Carbolic 10% in olive oil.
25. Carbolic-glycerine	1.9	2.5	2.0	1.4	Carbolic 12% in glycerine.
26. Metaphen tincture (1:200)	1.9	1.9	1.4	1.7	The anhydride of 4, nitro, 5-hydroxy-mercuri-orthocresol.
27. Metaphen tincture (1:2500)	0.5	0.8	0.3	0.5	The same as 26.
28. Phenol, 5% aq. sol.	1.0	1.0	1.0	1.0	Standard control titrated with bromine.

*N. Aspergillus niger. *W. Aspergillus wentii.
*G. Aspergillus glaucus. *F. Aspergillus fumigatus.

The use of Castellani's Solution was suggested because of its wide application in the treatment of tinea infections. Its composition* suggests an excellent germicide.

One of us, H. B. S., was struck by the fact that very few service station operatives have been observed to suffer from infections of the hand. We wondered if gasoline and the various naphtha compounds they handle might possess marked bactericidal properties. This and the fact that thymol was found to be soluble in them led to their comparison with other substances.

*Castellani's Solution consists of basic fuchsin gms. 1, boric acid, Sat. Aq. Sol., 1 cc., resorcinol, gms., 10, alcohol 95 per cent, 10 cc.

TABLE II

ACTION OF VARIOUS SUBSTANCES UPON SOME ASPERGILLI FOUND IN OTOMYCOSIS

ORGANISM Substance Employed	Phenol Ratio *N	*W	*G	*F	
1. Zinc oxide-boric	0.9	1.9	1.2	1.8	Boric acid, zinc oxide, eq. pts.
2. Alcohol-boric	0.9	1.5	0.5	1.5	Saturated alcoholic solution.
3. Phe-mer-nite, 1:1000 †G	0.8	0.9	0 7	1.0	Phenylmercuric nitrate solution.
4. Phe-mer-nite, 1:3000 †R	0.9	1.0	1.1	0.6	Phenylmercuric nitrate solution.
5. Phe-mer-nite, 1:2000 †G	0.6	0 8	0.4	0.5	Phenylmercuric nitrate solution.
6. Phe-mer-nite-ointment	0.5	0.6	0.7	0.5	Phenylmercuric nitrate ointment.
7. Whitfield's-alcohol	0.7	0.2	0.3	0.6	Whitfield's in 95% alcohol.
8. Thymol-yellow oxide of mercury	0.3	2.0	1.5	0.2	2% thymol in yellow oxide of Hg.
9. Mercurochrome, 2% aq	0.6	0 9	0.5	0.5	The disodium salt of 2:7 dibrom-4-hydroxymercuri-fluorescein.
10. Mercurochrome, 5% aq	0.9	1.0	0.7	1.0	
11. Methylene blue	0.9	1.3	0.6	1.1	Saturated alcoholic solution.
12. Silver nitrate-alcohol-glycerine	0.5	0.4	0.3	0.7	$AgNO_3$ grs. V in oz. III of alcohol-glycerine, eq. pts.
13. Silver nitrate aq	0.9	1.3	0.8	0.9	$AgNO_3$ grs. 100, aq. oz. I.
14. Mercury bichloride aq	0.23	0.25	0.2	0.23	$HgCl_2$ gr. 1, aq. oz. VI.
15. Hexylresorcinol solution	0.3	0.07	0.3	0.0	Hexylresorcinol 1:1000.
16. Fowler's solution	0.0	0.9	0.6	0.7	Potassium arsenite soln. (As_2O_3-1%).
17. Hydrogen peroxide	0.0	0.9	0 0	0.3	H_2O_2-3% solution.
18. Hydrogen peroxide alcohol	0.0	0.3	0.0	0.5	H_2O_2-3% soln. pts. 2 in alcohol 95%, pts. 1.
19. Listerine	0.0	0.2	0.2	0.0	Thymol content 0.075%.
20. Pepsodent antiseptic	0.0	0.3	0.1	0.2	Thymol as chlor-thymol 0.075%.
21. Magnesium oxide-salicylic acid	0.0	0.8	0.0	0.0	MgO-salicylic acid eq. pts.
22. Salicylic-alcohol	0.0	0.4	0.0	0.4	Salicylic acid 2% in alcohol 95%.
23. Phenol 5% aq. solution	1.0	1.0	1.0	1.0	Standard control titrated with bromine.

*N. Aspergillus niger.
*G. Aspergillus glaucus
*W. Aspergillus wentii.
*F. Aspergillus fumigatus.
†G. Green.
†R. Red.

"Pepsodent Antiseptic" and "Listerine" were tried as fungicidal agents merely because of the thymol they contain and which is in both preparations too small to be of fungicidal value.*

Pepsodent Antiseptic-Thymol as Chlorthymol	0.07 per cent
Listerine-Thymol	0.075 per cent

We have examined sixty-nine substances alone or in combination. In many instances where they proved worthy of trial from the standpoint of fungicidal or germicidal action they were used and their clinical application evaluated.

Tables 1 and 2 show the results obtained on each of the four separate species of aspergilli. With few exceptions these results are strikingly similar for each species of mold. It is well known that

*From figures kindly furnished by the Chemical Laboratory of the American Medical Association:

TABLE III

SUBSTANCES USED OR ADVOCATED FOR USE IN OTOMYCOSIS, THE FUNGICIDAL EFFECT OF WHICH HAS PROVEN NEGATIVE IN ALL INSTANCES WHERE TRIED BY THE CUP PLATE METHOD ON A. WENTII, NIGER, GLAUCUS AND FUMIGATUS RESPECTIVELY

1.	Alcohol, absolute.	
2.	Alcohol, 95%.	
3.	Alcohol, 70%.	
4.	Alcohol, 50%.	
5.	Alcohol, 40%.	
6.	Alcohol, 30%.	
7.	Copper, sulphate, 1:240.	
8.	Carbolic, aq. —	Carbolic, gtt. V. aq. oz. 2.
9.	Carbolic, aq. —	Carbolic, gtt. V. aq. oz. 1.
10.	Calcium hypochlorite, grs. 2.	Aq. oz. 1.
11.	Glycerine-aq.-zinc sulphate	Glycerine—H_2O eq. pts. oz. 1. $ZnSO_4$ grs. 2.
12.	Mercury Bichloride	$HgCl_2$ aq. 1:300,000.
13.	Potassium permanganate	$KMnO_4$ aq. 1:100.
14.	Potassium permanganate	$KMnO_4$ aq. 1:1000.
15.	Potassium permanganate	$KMnO_4$ gr. 1, aq. oz. 1.
16.	Silver nitrate	$AgNO_3$ aq. 1:16,000.
17.	Sodium thiosulphate	5% aq.
18.	Sodium thiosulphate	10% aq.
19.	Sodium thiosulphate	15% aq.
20.	Sodium salicylate	Sod. salicylate gr. 20, aq. oz. 1.
21.	Tannin-alcohol	Tannin gr. X, alcohol 40% oz. 1.
22.	Zinc sulphate, aq.	$ZnSO_4$ gr. 2, aq. oz. 1.
23.	Zinc sulphate, aq.	$ZnSO_4$ gr. 4, aq. oz. 1.
24.	Zinc sulphate, aq.	$ZnSO_4$ gr. 8, aq. oz. 1.
25.	Zinc sulphate-glycerine	$ZnSO_4$ gr. 2, glycerine oz. 1.

alcohol, at best, is recognized as a weak germicide, even in strengths of 50 to 70 per cent. In Table 1 it is interesting to note that alcohol has no fungicidal action in any of the strengths indicated. Duplicate and repeated tests on all four species always gave like results.

Table 3 contains substances, used alone or in combination, which showed no fungicidal action on all four species of aspergilli.

Because of the close agreement in fungicidal action of the various substances used upon each species of mold, we have felt justified in concluding that the fungicidal effect is comparatively the same upon all species of aspergilli. Hence genera only and not species need to be determined in the treatment of otomycosis.

Table 4 is based upon such conclusions. Figures under P. R. represent "Phenol Ratio" averages of results, indicated in Table 1 and 2, are arranged in descending order. Figures under R. indicate the relative order of effectiveness or "Relative Rank," compiled from

TABLE IV

ACTION OF VARIOUS SUBSTANCES UPON SOME ASPERGILLI FOUND IN OTOMYCOSIS

Averages of Organisms N, W, G, F, Arranged in Order of Effectiveness.

No.	Substance Employed	Pr.	R.
1.	Thymol-cresatin-alcohol	2.6	1
2.	Cresatin	2.55	2
3.	Thymol-merthiolate, 45, alcohol 95%	2.5	3
4.	Thymol-alcohol, 95%	2.5	3
5.	Castellani's solution	2.5	3
6.	Castellani's solution, thymol	2.43	4
7.	Thymol-alc., 70%	2.42	5
8.	Thymol, 2% aq. emulsion	2.1	6
9.	Merthiolate tincture, 1:1000	1.97	7
10.	Thymol-alcohol, 50%	1.95	8
11.	Merthiolate solution, 1:1000	1.95	8
12.	Carbolic, 12% in glycerine	1.95	8
13.	Thymol-gasoline	1.95	8
14.	Iodine tincture	1.88	9
15.	Carbolic, 10% in olive oil	1.82	10
16.	Metaphen tincture, 1:200	1.73	11
17.	Thymol-sodsol	1.55	12
18.	Zinc oxide-boric, eq. pts.	1.44	13
19.	Thymol-boric-iodide	1.4	14
20.	Alcohol-boric (Sat. solution)	1.1	15
21.	Thymol, 2% in yellow oxide of mercury	1.01	16
22.	Phenol	1.0	17
23.	Silver nitrate aq.	0.97	18
24.	Methylene blue, sat. alc.	0.96	19
25.	Phe-mer-nite, 1:3000-R.	0.91	20
26.	Sodsol (naphtha)	0.9	21
27.	Mercurochrome, 5% aq.	0.9	21
28.	Phe-mer-nite, 1:1000-G.	0.89	22
29.	Mercurochrome, 2% aq.	0.61	23
30.	Phe-mer-nite ointment	0.6	24
31.	Phe-mer-nite, 1:2000-G.	0.57	25
32.	Fowler's solution	0.55	26
33.	Tinct. metaphen, 1:2500	0.52	27
34.	Silver nitrate, glycerine, alcohol (95%)	0.47	28
35.	Whitfield's-alcohol (95%)	0.45	29
36.	H_2O_2, 3%	0.30	30
37.	Mercury bichloride, 0.04% aq.	0.23	31
38.	H_2O_2-alcohol, 2%, in alcohol 95%	0.2	32
39.	Salicylic, 2%, in alcohol 95%	0.2	32
40.	MgO-salicylic, eq. pts.	0.2	32
41.	Hexylresorcinol, S. T. 37	0.17	33
42.	Pepsodent antiseptic	0.15	34
43.	Listerine	0.1	35

R.—Red.
G.—Green.
N.—Aspergillus niger.
W.—Aspergillus wentii.
G.—Aspergillus glaucus.
F.—Aspergillus fumigatus.
Pr.—Phenol ratio.
R.—Relative rank.

TABLE V

ACTION OF VARIOUS SUBSTANCES UPON STAPHYLOCOCCUS AUREUS ARRANGED IN ORDER OF EFFECTIVENESS

No.	Substance Employed	Pr.	R.
1.	Tincture iodine	2.69	1
2.	Thymol-boric iodine	2.69	1
3.	Thymol-merthiolate, 45-alcohol 95%	2.07	2
4.	Merthiolate solution, 1:1000	1.95	3
5.	Merthiolate tincture, 1:1000	1.92	4
6.	Thymol-alcohol 70%	1.92	4
7.	Thymol-alcohol, 95%	1.92	4
8.	Hydrogen peroxide, 3%	1.78	5
9.	Zinc-oxide-boric, eq. pts.	1.78	5
10.	Castellani's thymol	1.76	6
11.	Castellani's solution	1.76	6
12.	Cresatin	1.74	7
13.	Tincture metaphen, 1:200	1.61	8
14.	Thymol-cresatin-alcohol	1.61	8
15.	Thymol alcohol, 50%	1.61	8
16.	Thymol, 2%, aq. eml.	1.53	9
17.	Mercurochrome, 5%, aq.	1.53	9
18.	Phe-mer-nite, 1:3000-R.	1.53	9
19.	Methylene blue sat. alc.	1.53	9
20.	Mercurochrome, 2%, aq.	1.44	10
21.	H_2O_2-2%, in 95% alcohol	1.37	11
22.	Phe-mer-nite, 1:1000-G.	1.30	12
23.	Phenol, 5%, aq.	1.00	13
24.	Thymol, 2%, in yellow oxide of mercury ointment	0.92	14
25.	Hexylresorcinol S. T. 37	0.92	14
26.	Metaphen, 1:2500	0.84	15
27.	Sodsol (naphtha)	0.84	15
28.	Thymol, 2%, in gasoline	0.84	15
29.	MgO-Salicylate, eq. pts.	0.76	16
30.	Phe-mer-nite, 1:2000-G.	0.76	16
31.	Carbolic, 12%, in glycerine	0.68	17
32.	$HgCl_2$-aqua-0.04%	0.53	18
33.	Silver nitrate, 20%, aq.	0.53	18
34.	Thymol, 2%, in sodsol	0.46	19
35.	Alcohol-boric (Sat.)	0.38	20
36.	Silver nitrate-glycerine-alcohol	0.30	21
37.	Carbolic, 10%, in olive oil	0.30	21
38.	Salicylate, 2%, in alcohol, 95%	0.30	21

values indicated under "P. R." Thus it is indicated that thymol-cresatin-alcohol ranks first as a fungicide, as indicated by the method employed. The results of our studies indicate that alcohol is non-effective as a fungicide. We use it in our combinations of thymol-cresatin as a solvent for thymol, a diluent for cresatin and for its drying effect in the ear. We hope to show by experimentation, now

being conducted, the minimum time in which thymol and various combinations of such will destroy aspergilli both on epithelial surfaces and in vitro.

Bearing in mind the probability of secondary invasion of pyogenic organisms, particularly staphylococci, we were interested to determine, by the same method of comparison employed for aspergilli, the germicidal action of the more important substances found to possess some degree of fungicidal action; especially where some of the well established germicides had been employed in combination with fungicidal agents.

Table 5 indicates the Phenol Ratio and the relative effectiveness of these substances on staphylococcus aureus and arranged similarly to their action on aspergilli as shown in Table 4.

Table 6 gives a comparison of the relative fungicidal and germicidal action of these substances.

In order to arrive at some evaluation of the combined fungicidal-germicidal action, averages of relative values, indicated in Table 6, have been calculated and arranged in relative descending order of effectiveness as shown in Table 7. Those values would presumably vary under actual conditions where secondary pyogenic invaders exist, the degree depending upon factors not easily evaluated in vitro. Clinical trial, however, in some 150 cases using 2 per cent thymol with 70 or 95 per cent alcohol as a solvent or in various combinations, such as cresatin and alcohol, merthiolate and alcohol, or as a powder for insufflation, with iodine and boric acid, has convinced us of its value as a fungicide and of the combined fungicidal-bactericidal activity in cases complicated by accompanying pyogenic organisms.

Methods of examination and treatment which have evolved from our findings and which have given excellent clinical results over a six-year period are enumerated below.

EXAMINATION

Culture is not necessary in many cases, as the condition may frequently be recognized from the appearance of the canal, especially if the magnifying lens of an otoscope is used or a plus 7 lens is held in front of the ear speculum. Microscopic examination of the scrapings applied to a glass slide in a drop of saline with a cover-slip applied and examined under the low power objective will often disclose the mycelial threads and conidia or fruiting bodies. Figs. 2 and 3.

TABLE VI

COMPARISON OF RELATIVE EFFECTIVENESS OF VARIOUS SUBSTANCES ON MOLDS (ASPERGILLI) AND S. AUREUS

Organisms No.	Substance Used	A.	S.
1.	Thymol-cresatin-alcohol	1	8
2.	Cresatin	2	7
3.	Thymol-merthiolate-alc.	3	2
4.	Thymol-alcohol, 95%	3	4
5.	Castellani's solution	3	6
6.	Castellani's thymol	4	6
7.	Thymol-alcohol, 70%	5	4
8.	Thymol aq. emulsion	6	9
9.	Merthiolate tinct., 1:1000	7	4
10.	Thymol-alcohol, 50%	8	8
11.	Merthiolate solution, 1:1000	8	3
12.	Thymol-gasoline	8	15
13.	Carbolic, 12%, in glycerine	8	17
14.	Tincture iodine	9	1
15.	Carbolic, 10%, in olive oil	10	21
16.	Tinct. metaphen, 1:200	11	8
17.	Thymol-sodsol	12	19
18.	Zinc oxide-boric	13	5
19.	Thymol-boric-iodide	14	1
20.	Alcohol-boric	15	20
21.	Thymol-yellow ox. of Hg.	16	14
22.	Silver nitrate, aq. 20%	17	18
23.	Phenol, 5%, aq.	17	13
24.	Methylene blue, sat. alc. solution	18	9
25.	Phe-mer-nite, 1:3000, red	19	9
26.	Mercurochrome, 5%, aq.	20	9
27.	Sodsol (naphtha)	20	15
28.	Phe-mer-nite, 1:1000, green	21	12
29.	Mercurochrome, 2%, aq.	22	10
30.	Phe-mer-nite, 1:2000 green	23	16
31.	Metaphen, 1:2500	24	15
32.	$AgNO_3$-glycerine-alcohol	25	21
33.	H_2O_2, 3%	26	5
34.	$HgCl_2$ aq., 0.04%	27	18
35.	H_2O_2 alcohol, 2%, in alcohol, 95%	28	11
36.	Salicylic acid, 2%, in alcohol, 95%	28	21
37.	MgO-salicylic, eq. pts.	28	16
38.	Hexylresorcinol	29	14

A—Aspergilli, computed from average of action of various substances on A. Niger, wentii, glaucus and fumigatus.

S—Staphylococcus, computed from average of action of the same substances on staphylococcus aureus.

TABLE VII

COMBINED EFFECT OF VARIOUS SUBSTANCES ON ASPERGILLI AND STAPHYLOCOCCUS AURIUS. COMPUTED FROM AVERAGES SHOWN IN TABLE VI

No.	Substance Used	Relative Value
1.	Thymol-merthiolate solution, No. 45, alcohol, 95%	1
2.	Thymol-alcohol, 95%	2
3.	Thymol-cresatin-alcohol, 95%	3
4.	Thymol-alcohol, 70%	3
5.	Castellani's solution	3
6.	Cresatin	3
7.	Castellani's thymol	4
8.	Tincture iodine	4
9.	Merthiolate solution No. 45, 1:1000	5
10.	Merthiolate tinct., 1:1000	5
11.	Thymol, 2%, aq. emulsion	6
12.	Thymol-boric-iodide	6
13.	Thymol alcohol, 50%	7
14.	ZnO-boric, eq. pts.	7
15.	Tinct. metaphen, 1:200	8
16.	Thymol-gasoline	9
17.	Carbolic, 12%, in glycerine	10
18.	Methylene blue, sat. alc. solution	11
19.	Phe-mer-nite, 1:3000-Red	12
20.	Mercurochrome, 5%, aq.	13
21.	Phenol, 5%	14
22.	Mercurochrome, 2%, aq.	14
23.	Thymol-yellow ox. hg.	14
24.	Hydrogen peroxide, 3%	15
25.	Carbolic, 10%, in olive oil	15
26.	Thymol sodsol	15
27.	Phe-mer-nite, 1:1000-Green	16
28.	Boric-alcohol, sat. solution	17
29.	Silver nitrate, 20%, aq. solution	17
30.	Sodsol (naphtha)	18
31.	Hydrogen peroxide, 2%, in 95% alcohol	19
32.	Metaphen, 1:2500 solution	19
33.	Phe-mer-nite, 1:2000-Green	19
34.	Hexylresorcinol, S. T. 37	20
35.	MgO-salicylic acid, eq. pts.	21
36.	$HgCl_2$, 0.04%, aq. solution	22
37.	$AgNO_3$-glycerine-alcohol, 95%	23
38.	Salicylic, 2%, in 95% alcohol	24

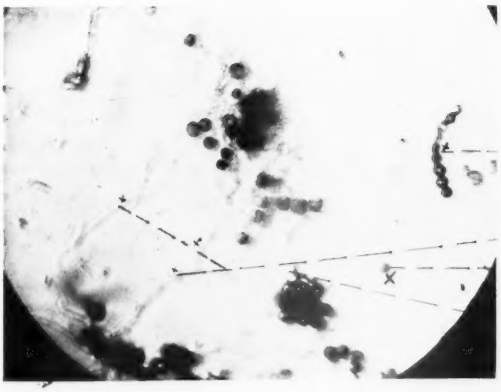

Fig. 2

Fig. 3

Figs. 2 and 3. Photomicrographs showing development of aspergillus wentii in hang-drop preparations x450.

Where culture is desired the ordinary Loeffler's blood serum tube, as used for diphtheria cultures, proves satisfactory. Molds will develop in two or three days at room temperature. Differentiation of species is not necessary, the most resistant being vulnerable to thymol in alcohol or mixtures of the same in the combinations listed.

TREATMENT

The chief object in the treatment is a thorough removal of the infected material, being especially careful of the acute angle of the tympanic membrane and the canal wall, followed by the application of a fungicide and germicide. In some cases one treatment may suffice, and most any effective germicide plus mechanical removal of the fungus will be all that is necessary to clear up the infection. Should the canal be filled with a dirty membraneous material, colored according to type of mold, with inflammation of the skin and neighboring structures, then more heroic measures are required. Hydrogen peroxide and irrigations will remove the loose débris, but the mopping with cotton tipped applicators moistened with a bland oil and a small hook or forceps must be used to remove this tenacious material from the skin and recesses without further traumatizing the tissues. After the ear is cleaned as thoroughly as possible, 2 per cent thymol in cresatin is applied with a cotton swab. Should there be much irritation this solution may be diluted with olive oil. A wick of cotton saturated in this oil is left in the canal until the next day or the thymol-cresatin-olive oil may be prescribed for home use, applied once daily with a medicine dropper. The number of office treatments, usually two or three, several days apart, and the type of "drops" for home use are suggested from the amount of inflammation present. In a very tender ear, bland substances must be used, but as this inflammation subsides more drying "drops" and office procedures are indicated, in which case alcohol mixtures or a powder of 2 per cent thymol, 2 per cent iodine, in boric acid* are used. Complications modify the line of treatment greatly, and because of the frequency with which mycosis occurs and the success attained with thymol we advocate its use in practically all office remedies. The 2 per cent strength is effective and causes no pain or irritation. The following combinations have proved satisfactory, depending upon the indication for their particular use:

*Powdered boric acid is used. Dissolve thymol and iodine in ether and add boric. Mix and allow ether to evaporate. Keeps best in glass-stoppered dark colored bottles. Dark bottles easily made by inserting an ordinary cork, tying a string around the neck, dipping in black enamel paint and drying at the end of string overnight.

(1) ℞ Thymol ..... grs. 10
Boric acid ..... grs. 10
Alcohol ..... oz. 1

(2) ℞ Thymol ..... grs. 10
Calot solution ..... oz. ½-1
(Especially indicated in chronic middle ears with granulations.)

(3) ℞ Thymol ..... grs. 5
Cresatin ..... oz. ½
(Dilute with alcohol or olive oil if desired.)

(4) ℞ Thymol ..... grs. 10
Merthiolate solution 1-1000
Alcohol ..... aa oz. ½

(5) ℞ Thymol ..... grs. 10
Tinct. merthiolate ..... oz. 1
(Color objectionable for home use)

(6) ℞ Thymol ..... grs. 10
Iodine ..... grs. 10
Boric acid qs. ..... oz. 1

Thymol, 2 per cent, in ammoniated mercury ointment, yellow oxide of mercury ointment, Whitfield's, merthiolated ointment, etc., may be used as desired.

COMMENT

Formulæ for the various combinations of substances are shown in Tables 1 and 2. In Table 3 are listed twenty-five chemicals, used alone or in combination, which have failed to show any degree of fungicidal action by the procedure outlined. Perhaps some of them were advocated for use as bactericidal agents. We have made no attempt to determine this in instances where fungicidal effect was nil. No doubt if stronger concentrations were used some of them might prove more effective.

In spite of the negative fungicidal action of alcohol it has proven valuable in otomycosis because of its cleansing and drying action, also as a solvent for the removal of accumulated cerumen and chemicals applied in treatment. In like manner the virtues attributed to the time honored use of salicylic acid in alcohol apparently arise from the exfoliation produced, since we find it occupying last place in combined fungicidal-germicidal action (Table 7).

Hydrogen peroxide, 3 per cent aqueous, occupies fifth place, Table 6, as a bactericide, and twenty-sixth place as a fungicide. Although a poor fungicide, it is a valuable adjunct as a preliminary cleansing agent, dissolving cerumen, crusts, scales and pus in addition to its bactericidal activity.

Boric-alcohol (boric, 4 per cent, in 95 per cent alcohol), long useful in a discharging ear, owes its action to bacteristatic rather than to bactericidal properties. Like others, it proves useful in certain cases of otomycosis, but alone would not prove as valuable as 2 per cent thymol in 95 per cent alcohol, which ranks second in combined action.

The use of phenol in glycerine is justified in many instances, especially in complicating acute otitis media; phenol, for its analgesic quality; glycerine, being somewhat hygroscopic, may cause an exosmotic current from the middle ear with lowering of pressure in the tympanic cavity.

The use of 2 per cent thymol in yellow oxide of mercury, recommended by us, seems contrary to our findings, since it ranks sixteenth as a fungicide and fourteenth as a bactericide. We feel that this method of testing ointments is not a fair one, since there is little chance of diffusion. By lightly streaking plates seeded with molds, and others with staphylococci, cultures failed to develop under these areas, but were profuse away from the clear zone of diffusion. Its action and that of thymol in ammoniated mercury, as applied in the ear, is one of direct contact and finds special use when applied to epidermal surfaces of the external meatus, especially where there is an accompanying dermatitis.

Thymol is only slightly soluble in water (0.088 gms. in 100 cc.), yet we find in Table 6 that 2 per cent thymol added to water and shaken well before testing so as to form a fine emulsion, is very effective as a fungicide and a germicide, occupying sixth place as a fungicide and ninth as a germicide against the test organism, placing it far ahead of thirty others formerly used, still used, or advocated for use as fungicides. As a germicide in the strength used it outranks some twenty-two other substances or combinations of such.

The positive action of the emulsion, the negative action of alcohol in all dilutions, Table 3, and the activity of thymol in 70 and 95 per cent alcohol, Table 4, is convincing evidence that the fungicidal action is due entirely to thymol and that alcohol acts only as solvent.

Castellani's solution without thymol appears more effective than when thymol is added, though the difference is slight. In the evaluation for molds, Table 4, the P. R. for Castellani's was 2.5 and with thymol added it was 2.42. For staphylococci (Table 5) the ratio was 1.76 in both instances. From this it may be concluded that where Castellani's solution is used the addition of thymol is not essen-

tial. Objection to Castellani's solution for home use may arise from the color, and also probable sensitiveness to phenol.

Choice of the most effective combinations, as indicated in Table 7, lies in the use of thymol with alcohol, 70 to 95 per cent, or thymol-alcohol mixtures with merthiolate or cresatin. These findings clinically applied have, in our estimation, established thymol as a valuable agent in the treatment of otomycosis.

SUMMARY

The fungicidal effect of sixty-nine substances, alone or in combination, has been compared in vitro and in a number of instances in vivo.

Similarly the bactericidal effect of thirty-eight of these substances has been studied and compared with the fungicidal.

As a result of these studies a system and formulæ for effective treatment of otomycosis are set forth.

Exclusive of twenty-four combinations giving negative results, an average of the effectiveness of eleven combinations containing 2 per cent thymol similarly compared with twenty-seven containing no thymol showed that the thymol combinations were 2.5 times more effective against aspergilli and 1.5 times more effective against staphylococci. Thymol combinations alone were 1.1 times more effective against aspergilli than against staphylococci.

CONCLUSIONS

Many of the so-called fungicidal substances used in the past and introduced for present use as such have no effect upon aspergilli. This is particularly true with alcohol in all dilutions.

The fungicidal effect of any effective substance is relatively the same for various species of aspergilli. Therefore the fungicide of choice will have a similar action on all species of aspergilli found in the ear.

Thymol, 2 per cent, in 70 to 95 per cent alcohol or combinations of thymol in alcohol, used with well established germicides, have proven to be most effective fungicides against aspergilli, as shown in vitro and in the treatment of some 150 cases of otomycosis.

The use of 2 per cent thymol in alcohol or in combination with effective germicides is more effective against aspergilli than non-thymol combinations.

The effective fungicides studied are likewise good germicides where staphylococci accompany otomycosis.

In the selection of any substance or combination for use in treatment on a basis of fungicidal or germicidal action or both, it is felt that substances having a phenol ratio less than one are of little value.

The value of the time honored use of alcohol, and of alcohol containing 2 per cent salicylic acid lies in the solvent and drying action of the former and in the exfoliation produced by the latter rather than in fungicidal-germicidal action.

Of the substances used in combination with 2 per cent thymol in alcohol, cresatin, merthiolate solution, No. 45 (1:1000), and thymol-boric-iodine mixture have yielded best results.

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LXXX

INTRALINGUAL THYROID: DISCUSSION WITH REPORT OF A LARGE STRUMA PRESENTING IN THE ANTERIOR PART OF THE TONGUE*

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BUFFALO

The term lingual thyroid refers to the presence of thyroid tissue in the tongue. Most lingual thyroids arise in the base of the tongue in the region of the foramen cecum. A smaller number push up into the tongue from a suprahyoid origin, coming to present above the frenum. These latter have been referred to by Kohl[12] as "tongue root goiter." A small number of cases with an associated "tongue root goiter" and "tongue base goiter" has been reported.

The following case of intralingual thyroid of the "tongue root" type is reported:

REPORT OF A CASE.

CASE 1.—In 1933, E. T., a colored housewife, age 69, an outpatient in the Buffalo City Hospital, complained of a swelling in the tongue which had manifested itself suddenly. At this time an examiner noted a "walnut-size tumor in the tip of the tongue." She entered the in-patient division of the hospital January 28, 1936, again complaining of a swelling in the tongue, and dysarthria and dyspnea of twelve and six months' duration respectively. She stated that the tongue swelling varied in size without apparent reason. She had borne six children. The menopause was reached ten years ago. The results of further enquiry were irrelevant.

The patient was a moderately obese, somewhat hebetudinous colored female. A convex swelling, the size of a large plum, bulged the under surface of the anterior part of the tongue forward, pushing the dorsum upward and backward. The mass on palpation seemed to be cystic and under tension. It did not pulsate, and no bruits could be heard over it. It was well circumscribed apparently and did not invade the floor of the mouth. There was no fixation of the tongue. One and one-half cubic centimeters of bloody fluid were aspirated from the tumor. The lingual duct orifices, foramen cecum and the frenum were normal in appearance. The mouth was edentulous.

The lateral lobes and isthmus of the thyroid were distinctly palpable in the usual position.

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*From the Department of Pathology, Buffalo City Hospital and the Medical School, University of Buffalo.

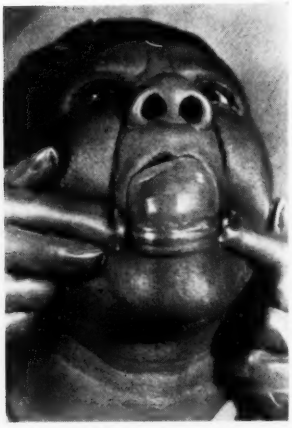

Fig. 1. A photograph of the tongue before operation. The tumor in the anterior part of the tongue is bulging forward, elevating and thinning its dorsum. Large veins are prominent over the convexity.

There were no signs of hypothyroidism. The blood pressure measured 146 mm. Hg. systolic and 74 diastolic. All other physical findings were unremarkable.

The red blood cells numbered 4,450,000 per cu. mm., the white cells 8,800; the blood hemoglobin was 70 per cent (Sahli). The differential white cell count, urinalysis and blood chemistry were normal. The blood Wassermann was negative.

Three days after admission, Doctors R. P. Dobbie and B. S. Custer incised the under surface of the tongue in the midline and easily extruded an encapsulated tumor. This wound healed uneventfully.

The patient remained in the hospital 32 days after operation. No symptoms or signs of hypothyroidism were manifested during this time. The basal metabolic rate fifteen days after operation was minus 8 per cent.

The tumor grossly measured 5.0 cm. x 3.7 cm. x 4.3 cm. It was roughly ovoid in outline, and possessed a thin fibrous capsule. On the cut surface fibrous septae arose from the capsule to course into the tumor proper, which was fleshy in consistency, had glistening points, and varied in color from a dead white to a pale pink-yellow. One cyst containing bloody fluid, measured 3.5 cm. x 2.5 cm. x 1 cm. Blood clot was adherent to its smooth shining wall. Other pin-head size points of hemorrhage were present in the fleshy portions.

Microscopically there were many polyhedral and cuboidal epithelial cells with large round darkly staining nuclei. Such cells were arranged in sheets, and as alveoli. In the latter the cells were sometimes several rows deep. The alveoli varied in size; most were small. Practically all were devoid of colloid. Mucicarmin stains were negative.

The stroma was scant, edematous and in some situations hemorrhagically infiltrated. The vessels were thin walled and large.

The diagnosis was fetal adenoma of thyroid.

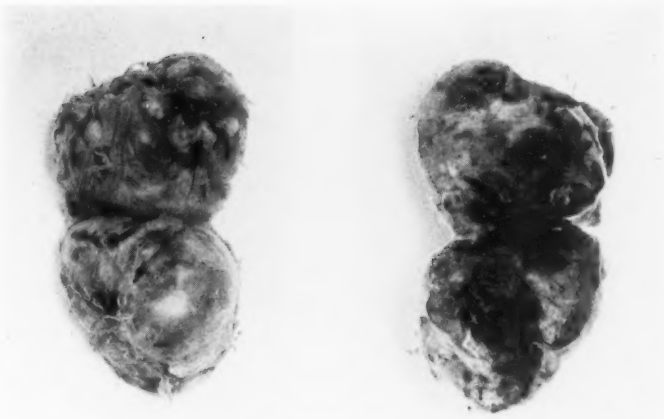

Fig. 2. Photograph of bisected tumor. The fibrous capsule over its convexity (left) and the cut surface (right) are depicted. The hemorrhagic cyst and a number of small points of hemorrhage can be seen. The structure grossly resembled thyroid tissue.

An analysis of a portion of the tumor was made by Mr. W. F. Millershoen, analytic chemist. His report was as follows: "The tissue when removed from the preserving fluid weighed 20.7 gm. A portion of it, 12.4 gm., moist weight, was dessicated in vacuo over sulphuric acid at room temperature to a constant weight of 2.466 gm. In two determinations of the dessicated gland no iodine was present by a method capable of detecting 0.01 mgm. The preserving fluid was likewise analyzed and no iodine found. The method of determination was a modification of that of E. Baumann: Ztschr. Physiol. Chem., 22, 1, 1897, and that of J. F. McClendon: Chemistry in Biology and Medicine."

Intralingual thyroids may be the result of arrested descent of the thyroid anlage, in which case they are designated heterotopic, or they may be the result of the development of aberrant rests, in which case they are termed accessory thyroids. In the heterotopic group of cases there is no thyroid in the cervical region.

The recorded incidence of lingual thyroids varies widely. In 1911, C. H. Mayo[17] stated that there were fifty recorded cases of lingual thyroid and added two others. Walther[26] found 70 described cases in 1912; Asch[1] in 1914 mentioned 95 instances, whilst, accord-

Fig. 3. Photomicrograph. Numerous small, and some large alveoli containing colloid, are seen. There are some solid sheets of epithelial cells. The thin walled, large sinusoids found elsewhere, are not seen in this particular field. Magnification x32.

ing to Hartley,[9] Lenormant could find only 43 cases in the literature in 1920. Kohl[12] knew of 93 cases in 1921; 83 of these were in females. Dore[6] analyzed 81 of 102 collected case reports in 1922. Grace and Weeks,[7] reporting a case in 1932, stated that there were 130 previously published cases, whereas Marchal, Soulie, Grupper and Roy[16] knew of only 32.

The relation of intralingual thyroids to other ectopic or aberrant thyroid is interesting. Leech, Smith and Clute[14] stated that only four cases of aberrant thyroid tumors were encountered in 4,000 instances of thyroid disease at the Lahey Clinic, whilst Cattell and Hoover,[5] from the same clinic, report that two cases of lingual thyroid were found in a total of 7,600 thyroid operations. In the University of Pennsylvania Hospital, Ulrich[25] found only two cases of intralingual thyroid in more than 4,000 patients with thyroid disease.

The embryology of the thyroid gland is thoroughly described by Grosser[8] and need not be mentioned here.

With rare exceptions, lingual thyroids originate in the base of the tongue, situated between the epiglottis and the circumvallate papillæ. Frequently they occupy the site of the foramen cecum. Sometimes they may appear to originate from one or the other side of it. This is due to arrest somewhat deeper in the tongue, with subsequent unilateral or asymmetric enlargement.

Lingual thyroids very rarely are found in the anterior part of the tongue as in the case herein reported. However, Kauffmann[11]

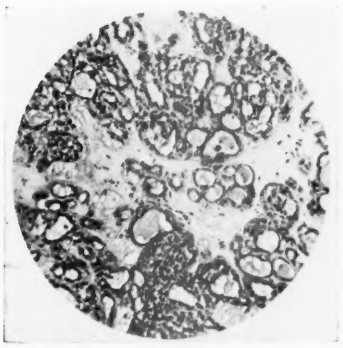

Fig. 4. Greater magnification, photomicrograph of section shown in Fig. 3. Note the alveoli containing colloid. Magnification x150.

cited Goris' patient, a fifty-year-old woman with a struma of hazelnut size in the tongue tip, and advanced the opinion of Erdheim that the anterior part of the tongue may harbor the median thyroid anlage. Nevertheless in the absence of any embryologic proof of such development, it seems more logical to assume that anteriorly situated lingual thyroids develop from the deep intralingual or suprahyoid arrest during descent of the thyroid anlage, with subsequent forward growth into the anterior part of the tongue.

Some reported "tongue base" thyroids appear to have been sessile with a flat disclike excrescence. Others present as an ovoid or round uniform swelling. They are usually smooth in outline, although the tumor reported by Bishop[3] was lobulated. A pedicle is uncommon. One lingual thyroid with a long free pedicle has been described by Harvey,[10] and one with a submucous pedicle extending to the foramen cecum, by Grace and Weeks.[7] The mucous membrane over lingual thyroids is apt to be thin and have many thin-walled large blood vessels which when traumatized may bleed.

Lingual thyroids vary in size. The smallest one reported is that of W. G. McCallum and M. Fabyan,[18] and the largest, measuring 8.5 cm. x 3.3 cm. x 2.3 cm., by Grace and Weeks.[7] Lahey[13] removed a lobulated, freely movable thyroid tumor the size of a hen's egg from the base of the tongue of a twenty-five-year-old female.

The greatest number of lingual thyroids have been reported in females. There seems to be no especial reason why a developmental anomaly should be confined to one sex. It is quite probable that lingual thyroids occur as commonly in males, but because of lack of

the various hormonal stimuli fail to enlarge sufficiently to cause symptoms. The fact that the incidence of reported cases of lingual thyroid in individuals under twelve years, a relatively low figure, is the same in both sexes appears to be additional evidence in favor of this explanation.

The morbid anatomy of lingual thyroids is the same as that of normally placed thyroid tissue. They are encapsulated by fibrous connective tissue. The cut surface grossly is fleshy, pink to red, and moist. According to the degree of physiologic activity, there may or may not be glistening flecks of colloid on the cut surface. There may be large or small cysts containing clear or hemorrhagic fluid bearing cholesterol crystals. As in cervical thyroids there may be multiple circumscribed adenomata. The histology may vary, as in cervical strumas, showing a colloid type, fetal adenoma, or simple hyperplasia. There may be much or little colloid. So far as I know, no one has studied the Golgi net of the epithelium of lingual thyroids.

The vascular network of the stroma may be rich or poor. The stroma proper may be greatly increased, as in the case of Grace and Weeks,[7] or slight in amount, as in the case of Marchal, Soulie, Grupper and Roy.[16] It may be hyalinized or calcified.

H. G. Wells,[28] who reported an autopsied case of dwarfism associated with lingual goiter and a cystic hypophysis, quoted six other cases of lingual thyroid associated with retarded growth. He considered the dwarfism in his case as strumiprivous in origin. The lingual thyroid contained 0.25 mgm. of iodine as compared with his established figure of 10 to 15 mgm. for a normal thyroid. This is the only published analysis of iodin content of a lingual thyroid that I have been able to find. Absence of iodine in the tumor in the present case may be due to several factors. Sollman[24] states that the iodine content of the thyroid gland is three times greater from June to November than from December to May. The patient in this case, well past the menopause, had a lingual thyroid removed in January, when, according to Sollman, the iodine content of the thyroid should have been near its low point. It is reasonable to assume that a very small amount of iodine in the tumor, just detectable in the summer months, might have escaped recognition in January. Furthermore, the histologic signs of activity in the tumor suggest that any iodine-fraction secretions would have been returned rapidly to the general circulation rather than remain stored in the gland. Determinations of iodine content of other lingual thyroids, with their comparison of the iodine of the blood and that of portions of the cervical thyroid may shed further light on this point.

Carcinoma of a lingual thyroid apparently is rare. Ashhurst and White[2] reported what they believed to be a carcinoma of a lingual thyroid. Their patient was a fifty-six year old male who had known of a mass to one side of the base of the tongue for twenty years. Removal, thirteen years before, was followed by swelling of the submaxillary glands. Because of dysphagia and pain in the tongue some of the mass, which measured 8 x 2½ cm., was removed. The histology of the tumor was that of "typical thyroid" with carcinoma in some sections. Stains for colloid or mucous were not reported. Improvement of symptoms followed. The final outcome was unknown.

Levi and Hankins[15] have recently reported a case of carcinoma developing in a thyroid at the base of the tongue. Hypothyroidism followed removal of the tumor.

Marchal, Soulie, Grupper and Roy[16] removed a fungating bleeding tumor from the right side of the tongue and also the left submaxillary glands of a fifty-nine-year-old man. At autopsy, two years later, metastases were found in the biceps muscles, the pleura and kidneys. The tumor was considered histologically to be thyroid with malignant neoplasia. Some alveoli contained colloid. No stain for mucus or colloid was reported. Brentano,[4] in 1911, reported a probable case of carcinoma of lingual thyroid with metastases to the regional lymph nodes. Kaufmann[11] states that "adenomas may spring from the mucous glands of the tongue. Not rarely the neoplastic gland cells are accompanied by a colloid transformation of the epithelium. This so-called colloid tumor may be multiple." In such cases as the above it is important to discriminate by special staining methods between a true thyroid carcinoma and a mucous gland carcinoma.

Symptoms of intralingual thyroid, when present, are those of any swelling in the tongue. These are enlargement and a sense of foreign body in the tongue, dysphagia, dysarthria, and if the tumor is large, dyspnea and attacks of asphyxia when in the dorsal prone position. Apparently the latter two symptoms are rarely found, for New[19] has stated that none of the ten adults with lingual thyroids, whom he examined, experienced pharyngeal obstruction at any time.

Hemorrhage, such as reported by Hartley,[9] and also by Pearlman,[20] is due to ulceration of the tumor or rupture of the large veins coursing over it.

Wegelin[27] has emphasized that accessory thyroids (those cases of intralingual thyroid in which there is also a cervical thyroid), as

well as heterotopic thyroids, are subject to the same physiologic stimuli as normally disposed thyroids. A history of enlargement of a tongue swelling with functional crests of the female endocrinology is almost pathognomonic of intralingual thyroid. There is an example of such changes in size in the case reported by H. Rubeli.[21] His patient was a woman whose lingual thyroid enlarged at puberty and fluctuated in size with the menstrual periods. During pregnancy it became so great as to produce obstructive symptoms which were relieved only by a tracheotomy. Following caesarean section the lingual tumor regressed to its former size. Enlargement at puberty of a lingual tumor, presumably thyroid, in a fourteen-year-old girl, has been reported by H. F. Ulrich[25] and also by J. Seldowitsch.[22] Ziegelman[29] described the case of a forty-eight year old woman, who first experienced obstructive symptoms of an intralingual thyroid at the onset of the menopause. However, such variations in size as these are not common.

Cretinoid characteristics reported in several instances of intralingual thyroid are an indication of lack of thyroid function and should not necessarily be interpreted as a part of the clinical pattern of intralingual thyroid.

The signs of thyroid tissue at the tongue base should not be confusing. A circumscribed red swelling between the angle of the circumvallate papillæ and the epiglottis, which may be sessile or pedunculated, sometimes bearing dilated vessels, can usually be assumed to be of thyroid tissue. In connection with this type of intralingual thyroid, Grace and Weeks[7] emphasize the possibility of pushing such a tumor from view into the pharynx when the tongue is depressed, and the importance of pulling the tongue forward to inspect its base.

An intralingual tumor which has originated in the suprahyoid region will be present, as in the case herein reported, in the ventral aspect of the tongue, lifting the dorsum high and thinning its mucosa.

Differential diagnosis of intralingual thyroid will include hemangioma, salivary cyst, dermoid cyst, ranula, papilloma, fibromyoma, and rarely metastatic tumors.

One can see no necessity for removal of a symptomless intralingual thyroid. Since some of the reported intralingual thyroid tumors in young individuals have been of the so-called "colloid goiter" type, it would seem reasonable to administer iodine to such patients with the hope that if it be of such type it might be reduced

in size. When obstructive symptoms develop, surgery is indicated. In view of the fact that an intralingual tumor may be heterotopic thyroid, and therefore the only thyroid tissue an individual may possess, it would seem logical, before removing it, to surgically explore the neck for thyroid, if the gland is not unequivocally palpable there. This is emphasized by Grace and Weeks[7] and also by H. F. Ulrich.[25] If such precautions are not taken, one may err in removing the sole thyroid tissue of an individual, with a subsequent cachexia strumipriva. Such has been reported a number of times. Spencer[23] mentions this complication in the cases of Ungermann, V. Chamisso de Boncourt, Meixner and Fletcher among others, and advises against removal of intralingual thyroids. He suggests that a wedge be removed from such tumors, if they should be so large as to give rise to obstructive symptoms.

SUMMARY

1. A case of the "tongue root" type of intralingual thyroid presenting far anteriorly in the tongue has been reported, together with the results of its assay for iodine and a detailed description of its gross and microscopic pathology.

2. The incidence, pathology, signs and symptoms, complications and treatment of intralingual thyroids have been discussed in relation to some of the pertinent literature.

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LXXXI

ANOMALY OF THE LATERAL SINUS: REPORT OF A CASE*

GORDON D. HOOPLE, M.D.

SYRACUSE

Anomalies of the lateral sinus have attracted the attention of embryologists and anatomists through the years. Because of its rarity, the clinical aspect of this condition has not been stressed, for the great majority of the reports in the literature are of specimens from the dissecting room. Surgical accidents account for most of the clinical cases.

To understand the ease with which anomalies may occur, a study of the embryology of the dural sinuses is in order. A detailed account of the development of the veins of the head is too intricate for present purposes. A summary, however, of the changing conditions which eventuate in the adult lateral sinus will be given to bring out the possibility of variations in the character and position of this structure.

Early in the development of the human embryo there is formed the primary head vein. Capillaries from adjacent tissues collect to form three dural plexuses which drain into the primary head vein. These are the anterior, middle and posterior dural plexuses. The otic vesicle, an early and important structure in the development of the human embryo, lies between the middle and posterior dural plexuses. The developing veins, in finding their ultimate location, are influenced by the presence of the vesicle. A second factor which influences greatly the course of the dural veins is the changes involved in the growth and development of the brain itself.

Streeter[1] gives three methods by which the course of the dural vessels change. First, a passive migration, where there is change in the position of the vein wall itself, due to some flexion or traction force acting upon it. Second, a spontaneous migration, in which there is a change in position of the blood stream only, where by a circumfluent anastomosis, the blood stream develops a new channel in the adjacent loops of the plexus with a corresponding

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dwindling of the previously used channel. Third, replacement channels differing from passive migration only in that it is not a gradual and progressive change, but an abrupt and immediately complete one.

When the human embryo increases in size to about 18 mm., owing to the growth of the middle ear and the labyrinth, the course of the primary head vein, ventrolateral to the otic capsule, becomes an unfavorable one and this portion becomes obliterated. Two new channels form, which compensate for this change. First, there is an anastomosis channel between the middle and posterior dural plexuses, which is dorsolateral to the otic capsule (Fig. 1.). Second, there is some fusion of the anterior and middle dural plexuses and the anterior plexus which formerly drained into the primary head vein, now drains caudally through this fusion and through the new channel between the middle and posterior dural plexuses. There is thus formed a new course for the flow of blood from before backwards as far as the jugular foramen, where it is continuous with the internal jugular vein (Fig. 2). This new course eventually becomes the lateral sinus. It should be noted that in this stage the course of the channels is horizontal but the flow of blood is from anterior to posterior, the reverse of the adult condition. The remaining portion of the primary head vein in this stage of the development of the embryo, aside from its cardinal portion, or internal jugular vein, is only that part in the region of the trigeminal nerve which soon becomes the cavernous sinus. This portion drains upward through the original trunk of the middle plexus, which is now the superior petrosal sinus, into the newly formed "lateral sinus." The part of the "lateral sinus" which lies between the superior petrosal sinus and the jugular foramen becomes the "sigmoid" portion. Anteriorly, the part which is made by the fusion of the anterior and middle plexuses becomes the "transverse" portion. "Thus in the 21 mm. embryo the dural channels in the region of the temporal bone have acquired essentially all their permanent connections, with the exception of the inferior petrosal sinus, which appears a little later." (Streeter.)

In embryos between 35 and 50 mm. long, because of the backward growth of the cerebral hemispheres, the newly formed chain of vessels which is to become the lateral sinus is carried backward till the anterior plexus is superior to the site of the middle plexus, which in turn lies above the posterior plexus. Except for the "sigmoid" portion the "transverse sinus" forms a straight line with the interior jugular vein and this line runs from above downward (Fig. 3). Between the 50 mm. embryo and adult life a further change takes place with the continued posterior growth of the cerebral hemispheres.

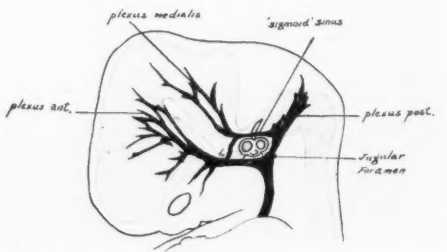

Fig. 1. Embryo 18 mm. Shows the fusion between the middle and posterior dural plexuses. (After Streeter.)

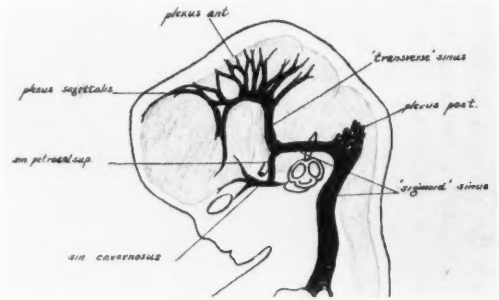

Fig. 2. Embryo 21 mm. Shows anterior plexus fused with the middle plexus. This forms the "transverse" sinus. The anterior plexus remains and shows the beginning of the sagittal plexus (After Streeter.)

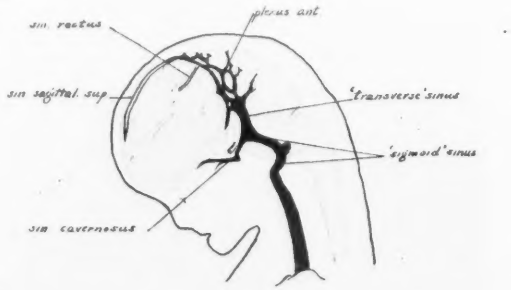

Fig. 3. Embryo 35 mm. Shows the "transverse" sinus and the internal jugular in a direct line from above downward. The superior sagittal sinus and the rectus sinus have formed. (After Streeter.)

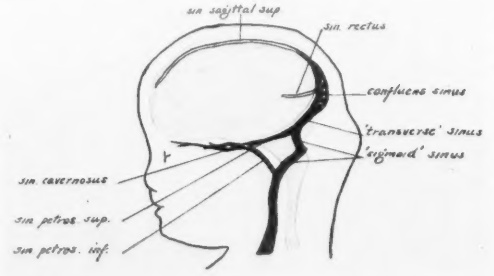

Fig. 4. Embryo 80 mm. Approaching the adult form. The "transverse" sinus is nearly horizontal and with the continued growth backward of the cerebral hemispheres, will become more so. (After Streeter.)

The "transverse sinus" bends backward till it reaches a horizontal position and is approximately at a 90° angle with the internal jugular vein (Fig. 4). In the earlier stages these changes just mentioned are carried out by spontaneous migration, while the channels for blood flow are still plexiform. Later, as the position and character of the vessels assume the adult form, the change is brought about by passive migration. In the final swing backward of the "transverse sinus," what remains of the anterior dural plexus comes to rest at the torcular herophili.

The superior sagittal sinus and the straight sinus are related to these findings because they have their origin from the anterior dural plexus. A sagittal plexus is formed from the ventral branches of the anterior plexuses of both sides. Eventually a single vessel is formed from the sagittal plexus, which is carried backward as the cerebral hemispheres enlarge posteriorly. The change from the sagittal plexus to the "sagittal sinus" takes place first in the ventral portion. The caudal fusion takes place last as the adult stage is reached. The formation of the "straight sinus" follows essentially that of the "sagittal sinus." As both of these sinuses have their origin from the anterior dural plexus, and both are single midline vessels, the course which they will take posteriorly when their blood flows into the two "transverse sinuses" (which are also anterior plexus in origin), is not always constant. The usual arrangement is for the superior sagittal sinus to empty into the right transverse sinus and the straight sinus into the left. Streeter says this occurs in approximately 90 per cent of the skulls he examined. Poirier[2] states this arrangement is present in about 50 per cent. Henrici and Kikuchi[3] reported the examination of the torcular region in thirty-five heads. They found eight different arrangements amongst the thirty-five specimens. Inasmuch as the superior longitudinal sinus may be completely absent (Portal has reported two cases), it may be divided into channels, or may take such an unusual course as to continue its route past the torcular and empty into the jugular bulb, it may be seen that there can be marked variation in the size of the two transverse sinuses. When there is great variation in the size or course of the superior sagittal or straight sinus there is usually a corresponding change in the size of the transverse sinus.

There are many other possibilities for change in the position and shape of the lateral sinus than have been mentioned above. Only the main connections and factors in the formation of the sinus have been given. This has been done to stress the fact that very few veins in the body have as much chance to be anomalous as does the lateral sinus.

During its formation, from three separate plexuses, it changes its direction from an anterior-posterior flow to one in the opposite direction. It turns on itself 180 degrees. In the adult stage it depends for its start at the torcular on an arrangement which is plexiform in origin and often maintains this characteristic in its final form. In fact, the plexiform origin of the lateral sinus itself sometimes enters into its final form. That such a small number of anomalies of the lateral sinus have been reported is surprising when these facts are considered.

REPORTED ANOMALIES OF THE LATERAL SINUS

Streit[4] has mentioned some anomalies of the lateral sinus which to some extent classify them. First, division of the transverse portion with a horizontal lamella, or a division into two separate channels. These same findings may be found in the sigmoid portion only. Second, absence of the transverse portion, with the descending portion starting at the entrance of the superior petrosal sinus. Third, exit of most of the blood stream from the transverse portion through the mastoid foramen with the jugular foramen markedly contracted. Fourth, absence of the sigmoid portion with exit of the blood through the mastoid foramen and into the petrosquamosal sinus in the mid-cranial fossa. To these anomalies should be added a fifth group in which the normal course is pursued, but there are variations in the size of the sinus. The changes in these instances are often dependent upon anomalies found in the contributing sinuses—the superior sagittal, straight, etc. Many of the reported cases of absence of or changes in the lateral sinus must be relegated to this last group for want of detail in description of the course of the vessel. Many of these note a difference in the size of the sinus on the two sides. Circulatory changes most frequently call attention to this condition. Eagleton[5] reported a case of sinus thrombosis in which the right (unligated) side was one-half the thrombosed left (ligated). Immediate choked disc and blindness followed ligation, together with involuntary disturbances, cyanosis, dyspnea, coma and finally death, which he believed was due to venous congestion. Rohrback[6] and Linser[7] have reported similar cases. Both terminated fatally. In Rohrback's case the lumen of the transverse and jugular veins, on the unligated side, were the size of a needle and quill respectively. In Linser's case, the ligated jugular was ten times the size of the unligated. G. Fisher, Asums, Berard, Krummer, Grunerts, Hölscher, Jensen, Dangel, Richards, Dench, Pansen and Streit have reported circulatory disturbances which demonstrated a likelihood that the other lateral sinus and jugular were too small to furnish a sufficient flow of blood from the

head. Swift[8] reported three cases with variations from the normal in the size of the lateral sinus. His third case is of particular interest. It was a new born child who died in status epilepticus on the morning of the third day after birth. Autopsy revealed extensive hemorrhages in the subdural spaces. The trabeculæ, which usually disappear in the transverse sinus, were still present, the rudiments of embryologic development. The jugular foramina were both rudimentary in character and the intracranial venous pressure was great enough to spread the newly formed venous sinuses and widely separate the two layers of dura. This apparently is the only reported case of bilateral absence of the lateral sinus.

Double lateral sinus has been reported by several observers. Brown[9] has reported a double sigmoid sinus. There were two channels for a distance of 4 cm. They were separated by a ridge of bone. Knott, Sperino, Streit, Hallett, Meckle, Dumont, Heine and Beyer have reported cases of double lateral sinus. From the embryologic viewpoint, these form in one of two ways: either the middle portion of the primary head vein persists to form the extra channel, or there is incomplete fusion into one channel of the many vessels which make up the blood pathway, between the anterior, middle and posterior dural plexuses in early foetal life. H. G. Tobey has recited, by personal communication, a case of double sigmoid sinus.

No report of a case of absence of the transverse portion with the start of the descending portion at the entrance of the superior petrosal sinus could be found. As several authors mention this possibility, there undoubtedly have been some cases recorded. Failure of the development of the original "transverse sinus" and enlargement of the connection between the middle dural plexus and the primary head vein (the superior petrosal sinus), would account for this anomaly.

Many cases of contracted jugular foramina have been reported. In most of these cases the sigmoid portion of the lateral sinus being small, the blood from the transverse portion drains through the mastoid foramen. Linser, in the examination of 1,022 skulls, found 29, or 3 per cent, in which the jugular foramen on one side was so contracted that the vein, in passing through, could not have been more than three or four mm. in diameter. The opposite side was three to four times as large. Of the 3 per cent, 2.5 per cent occurred on the left side and only 0.5 per cent on the right. Malacre described a skull in which the horizontal portions of both lateral sinuses were normal in size, but both passed out of the skull through enlarged mastoid foramina. Budde described a case in which both jugular

foramina were very small, and the right mastoid foramen was 1 cm. wide. In these cases, with a small descending portion of the lateral sinus, there is often found present a petrosquamosal sinus. This commences at the upper end of the sigmoid sulcus and courses anteriorly to the midcranial fossa, forming a deep groove in the temporal bone in the angle between the squamous and petrous portions. It leaves the skull through the spurious jugular foramen of Luschka, or through the foramen spinosum by way of the middle meningeal vein. Laff[10] reported three specimens which were shown him by Professor Alexander, with this sinus present. Zuckerkandl described a skull in which the petrosquamous sinus was 5.5 mm. wide. The sigmoid portion of the lateral sinus was present in these four specimens. Streit has reported two cases of petrosquamous sinus in which the sigmoid was present. Small sigmoid portions and absence of the sigmoid portion (see below) would derive embryologically from poor development of the blood channel between the middle and posterior dural plexuses.

Not many cases of absence of the sigmoid portion of the lateral sinus have been reported. Barkow[11] reported a case in which there was a large right jugular foramen, a much smaller jugular foramen on the left and a sigmoid fossa on the left, ending in an unusually large mastoid foramen, whereas, for a stretch of 20.9 mm., between the mastoid foramen and the left jugular fossa, the sigmoid sinus was entirely lacking. Hochstetter discovered such a specimen and this was reported by Laff. Macewen apparently reported an absence of the sigmoid portion when he cited a case in which there was absence of the lateral sinus with the blood passing to the petrosquamosal sinus and thus to the external jugular vein. Williams[12] reported a case of absence of the sigmoid sinus, apparently the first to be discovered at operation. On attempting to delineate the sigmoid during his operation, he came to the medial wall of the mastoid without encountering it. There was a large mastoid foramen and a large vein coming from the position of the knee of the sigmoid sinus, emptied through the foramen.

The cases cited above do not include all of the reported irregularities of the lateral sinus, but they illustrate the various types of anomalies of this structure. No mention of the part played by the occipital and superior petrosal sinuses has been made, though they change the course and character of the lateral sinus at times. In fact, there are so many factors which may influence this structure and so many combinations of these factors may be present, it seems strange that anomalies are not more frequent.

TOBEY-AYER TEST

This test has established itself as one to be included amongst the diagnostic procedures of the otologist. It is not infallible. No such claim has been made for it. Tobey and Ayer[13] called attention, in their original article, to some possible causes of failure of the test. One of these was variations in the size and character of the lateral sinus. A true anomaly of this sinus is, of course, relatively rare. Changes in the size of the lumen, however, while not common, are more frequently seen. Possible variations of spinal fluid pressure, because of anomalies and variations, should be borne in mind. The Tobey-Ayer test is of interest in the following case.

REPORT OF A CASE

CASE 1.—A boy, age 10, stricken with a severe attack of scarlet fever, had a right acute otitis media on the second day of his illness. A paracentesis was done. Temperature was irregular and ranged from 101 to 103 and 104 degrees daily. Postaural tenderness was present, together with marked swelling and tenderness of the anterior and posterior cervical glands on the right side. The condition remained the same for five days. Then the postaural tenderness and the gland swelling improved. The child appeared severely toxic, however, and the temperature remained high. Although there had been no chills, a blood culture was taken on the eighth day. On the ninth day there was a slight chill. On the following day the patient had a chill lasting for twenty minutes. A Tobey-Ayer test was done, with the following findings:

Pressure on the right jugular: no rise.
Pressure on the left jugular: rise from 100 to 350 cm.
Bilateral compression: rise to 400 mm.
Release of right jugular: no fall.
Release of left jugular: fall to 150 mm.

As a result of these findings, despite negative blood cultures (which remained so to the end), operation was ordered.

At operation an unusually large emissary vein was encountered, which was torn and bled profusely. When it was controlled and the region of the sigmoid portion of the lateral sinus was uncovered, no trace of the sigmoid portion could be found. The transverse portion was present to the knee (though much smaller than usual), and drained into the mastoid emissary.

The patient remained critically ill following the mastoidectomy. Signs of meningitis developed on the second day after the operation and death followed on the fourth day.

An autopsy was done. A purulent meningitis was present throughout. The transverse portion of the right lateral sinus was much smaller than the one on the left. It was about 3 mm. in diameter. It appeared to end blindly just below the knee, but on the outside of the skull it was seen to be continuous with the mastoid emissary. There was a large plexus of veins in the floor of the midcranial fossa and what appeared to be a petrosquamosal sinus connected the transverse portion with this plexus. The superior petrosal sinus was present and emptied into the transverse portion at the knee. The jugular foramen was not measured. The findings on the left side were apparently normal (Fig. 5).

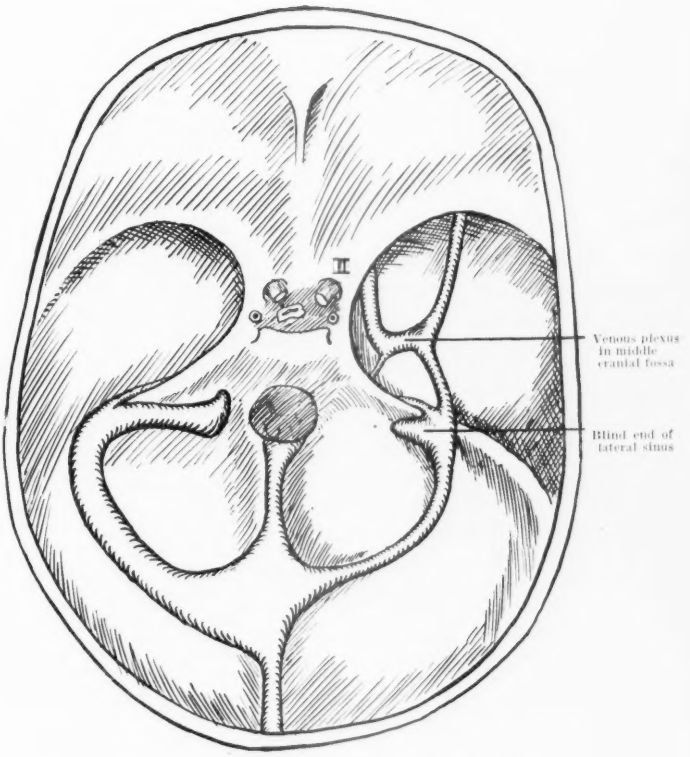

Fig. 5. Findings at autopsy in the case presented.

COMMENT

This patient presented a typical picture of lateral sinus thrombosis. The absence of the sigmoid portion furnished the condition which gave the readings of the Tobey-Ayer test the appearance of a complete thrombosis. It is interesting that the right mastoid was the one which was involved and the unusual vein findings were on this side.

SUMMARY

1. A review of the embryology of the lateral sinus is given, with some stress on the factors which may be the cause of anomalies.

2. The various types of anomaly of the sinus are mentioned, together with reported cases.

3. A case of absence of the sigmoid portion of the lateral sinus is presented.

Medical Arts Building.

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LXXXII

DIFFERENCES IN LOUDNESS RESPONSE OF THE NORMAL AND HARD-OF-HEARING EAR AT INTENSITY LEVELS SLIGHTLY ABOVE THE THRESHOLD*

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Performance of a considerable number of hearing tests in a quiet room by means of the Western Electric No. 2-A audiometer resulted in an impression that the response of the hard-of-hearing ear to sound intensity levels near the raised threshold of audibility differed somewhat from the response of normal ears near the normal threshold. The threshold values of the abnormal ears, with the possible exception of those affected with tinnitus, seemed to be more definite, critical or exact. It was believed for some time that this observation was due to the fact that the hard-of-hearing ears were further insulated from extraneous masking room and body noises by an attenuation equivalent to their hearing losses.

Additional observation, however, demonstrated that this difference in the response of the normal and the abnormal ears to intensity levels near threshold was not due primarily to differences in the noise levels experienced during the testing procedure. It was found that if an individual had a marked hearing loss, e. g., 85 db at 1024 cycles, he would describe the magnitude of the intensity of a level 95 db above the normal threshold (only 10 db above his threshold) as "loud." A sound 10 db above the threshold of the normal ear in a soundproof room, while readily perceived, is very "soft." A closely related phenomenon also was noted in relation to the intensity level preferred by individuals using vacuum tube hearing aids. It was found that the amplification or intensity level for speech reproduced in headphones which was preferred by the normal ear was in some instances approximately equal to the level also preferred by hard-of-hearing ears which had losses not exceeding 40 db over the frequency range from 64 to 8,192 cycles. In other words, ears with the hearing loss described above did not wish speech amplified as many

*From the Department of Otolaryngology, University Hospital, Iowa City, Iowa. Read at the meeting of the Acoustical Society of America, New York, April, 1935.

db above the threshold as did the normal ear. These observations were responsible for the impression that sounds a given number of db above the threshold of the hard-of-hearing ear are louder to that ear than the same sounds the same number of db above the threshold of the normal ear.

A review of the experimental literature revealed three investigations into the relation between intensity and loudness* in the hard-of-hearing ear which were significant in relation to the present study. Pohlman and Kranz,[8] as a result of work with a continuously variable frequency audio-oscillator on "Binaural Audition in a Subject With Ranges of Deficient Acuity," observed a phenomenon which induced them to ask the following question:

". . . in passing over a frequency range, will a portion of this range which shows a low value of acuity when at or very close to the low limit of audibility, continue to be noticeably low in comparison with the remainder of the range as the intensity of stimulus for the whole range is increased?"

They found by trial that:

". . . when the intensity of the stimulus was about ten times that required to hear the weakest part of the range, this low sensitivity portion was no longer noticeable as the frequency was varied to pass through it."

In the conclusion of this paper it was stated:

"Decreased acuity of hearing for certain limited portions of the tone scale, as determined by minimum audible sounds, is not a criterion of deafness with respect to ordinary intensities. Once the threshold of such a range of decreased acuity is attained, it takes comparatively little additional energy to cause the deficiency to seemingly disappear quite completely."

The interpretation of this conclusion may be clarified by the following hypothetical example: Suppose an individual has a normal threshold over the entire audible frequency spectrum except for a range of decreased acuity (a partial tone "gap") between 2000 and 4000 cycles, reaching a maximum equivalent to a 30 db loss at 3000 cycles. Now if he be stimulated with a continuously variable frequency over the audible range at an intensity level of 10 db or 20 db above the normal threshold of audibility, he will hear no sound at the frequency levels near 3000 cycles and therefore will be acutely conscious of the gap. At an intensity level slightly above 30 db he will be aware of a range of decreased acuity near 3000 cycles, since he will hear these frequencies very softly and will hear the adjacent higher and lower frequency levels much louder. But at an intensity level 40 db above threshold (10 db above the 3000 cycle "gap") the

*The term intensity refers to the physical energy in the sound wave; loudness refers to the subjective response or magnitude of the auditory sensation experienced.

ear would not be conscious at all of the area of "deficient acuity" which was apparent at the lower intensity levels.

Fowler[2] noted in an audiometric examination of a patient with a normal left ear but with a right ear with a loss of approximately 40 db near 4000 cycles that

"The threshold of hearing in the left ear was the same as that in all normal ears—a little indefinite without repeated search—whereas in the right ear (near 4000) there was no hesitancy in the sensing of the exact point every time it was reached; in other words, the sound was definitely absent or very apparent to the observer. The threshold appeared suddenly.

"Comparative loudness showed that the loudness of tones (near 4096) of from 10 to 25 sensation units above the threshold in the left (nonaffected) ear appeared the same loudness as 5 sensation units above the threshold in the right (affected) ear."

Guttman and Ham[3,4,5,6] also published a series of four articles which show that the normal and the hard-of-hearing ear respond differently to intensity increases under certain conditions. These experiments were designed to investigate masking phenomena in deafened ears. The following procedure was employed: The threshold intensity was obtained for a given tone, which was then increased 50 db above the threshold value. This tone, 50 db above threshold, was called the interfering tone. The threshold intensity of a second tone, called the primary tone, was obtained in the absence and then the presence of the interfering tone. The difference between the two threshold intensity values of the primary tone in the presence and absence of the interfering tone was called the differential threshold. As a result of their earlier experimentation they concluded that

". . . a given relative increase of energy produces in a deafened ear a greater hearing sensation than in a normal ear in the presence of an interfering tone whose multiple energy value above threshold is the same for every person."[3]

After refinement of technic and further work they summarized their findings as follows:

"1. The differential threshold (S_1-S_0) is less in a deafened than in a normal ear in the presence of an interfering or masking tone of the same energy level above threshold (50 db). The threshold refers to the one examined in each case. 2. The above finding is true in the so-called conduction as well as in perception deafness. In conduction deafness the above mentioned finding may be more pronounced. 3. The differential thresholds become smaller with increasing deafness."[4]

The conclusions of Guttman and Ham relative to the loudness of sounds a given number of db above the threshold of the hard-of-hearing ear quite properly are restricted to the condition in which a second interfering or masking tone is present. The observations of Pohlman and Kranz,[8] Fowler[2] and the writer indicate that sounds

a given number of db above the threshold of the hard-of-hearing ear are louder to that ear than the same sounds the same number of db above the threshold of the normal ear in a noise-free environment.

How can the validity of this observation be tested and exact quantitative data obtained upon the magnitude of the subjective response of the hard-of-hearing ear to intensity increments above threshold which can be compared directly with similar data obtained from the normal ear? The method employed by Fowler[2]—that of loudness comparisons by an individual with one normal ear and one hard-of-hearing ear gives comparable data, provided the difference in hearing loss between the two ears does not exceed about 50 db. (Certain frequencies having an intensity in excess of approximately 50 db above the normal threshold value in an ear phone held against the ear may be heard in the opposite ear, either by air conduction or bone conduction or both, if the opposite ear has normal acuity by air or bone conduction.)

An additional approach to the problem was suggested to the writer by the work previously completed on equal loudness level comparisons by the normal ear,[7 1] in which it was found that the ear is able to compare and equate quite accurately the loudness of two pure tones which differ in frequency. Application of the results of this work and the methods employed was made to ears with the clinical diagnosis of nerve deafness. Some such ears have practically normal acuity for the lower frequencies, but markedly raised thresholds for the higher frequencies. It was apparent that equal loudness level judgments by ears with this type of threshold also would provide a direct means of comparison of the loudness level resulting from increasing the intensity of different frequencies a given number of db above the normal and the raised threshold.

Kingsbury[7] and Fletcher and Munson[1] have contributed data, summarized in Fig. 1, which show various intensity levels above that of the normal threshold of audibility at which different frequencies are judged equally loud by the normal ear.

Reference to Fig. 1 shows that over the frequency range from 1000 to 4000 cycles the intensity levels of the Fletcher-Munson equal loudness level* contours are practically parallel with the intensity level of the threshold of audibility. That is, a frequency of 4000

*The intensity level of a sound is the number of db above the reference intensity. The loudness level of a sound is the intensity level of the equally loud reference tone.[1]

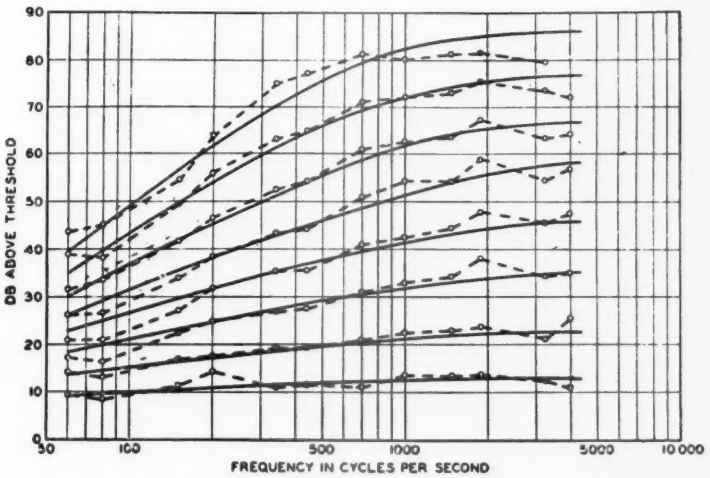

Fig. 1. Equal loudness level contours of pure tones.
Kingsbury's data - -
Fletcher-Munson data————

cycles 12 db above threshold is equally loud as frequencies of 3000, 2000 and 1000 cycles also 12 db above the threshold. The widest divergence in intensity of any shown equal loudness contour from the threshold value as a base line over this frequency range does not exceed approximately 6 db, occurring between the 50-60 db above threshold abscissae.

Fig. 2 shows comparable data obtained in a relatively quiet room by a modification of the technics employed in obtaining the data of Fig. 1.

Since this modification was necessitated by the apparatus available, and was used throughout the present study, a brief description of the technic employed will be given.

The apparatus consisted of two Western Electric No. 2-A audiometers, a headphone supplied with one of the audiometers, a headband and a double-pole double-throw switch. The same headphone was used throughout and was held in close contact with the ear by means of the headband. The threshold intensity level was obtained for each ear of the observer by means of one of the audiometers before beginning experimentation. A given tone was then produced in the headphone from audiometer *A* at, e. g., 4096 cycles

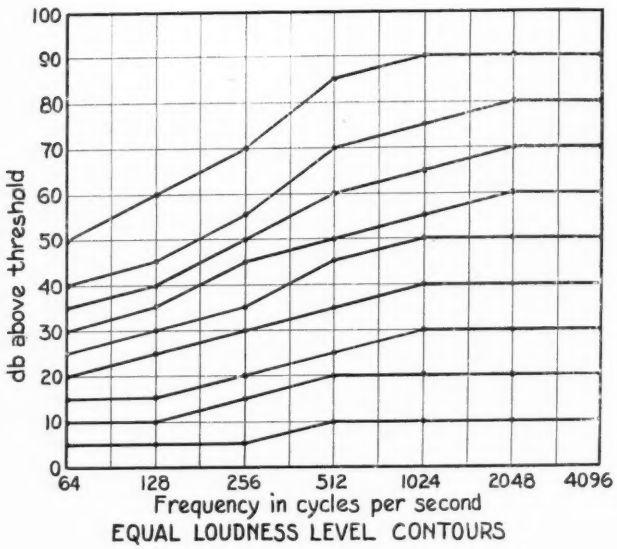

Fig. 2. Equal loudness level contours of pure tones. Writer's data.

at an intensity level of 10 db above the threshold intensity in the ear under test. Audiometer *B* was then adjusted to produce a frequency of 2048 cycles. The experimenter introduced these two frequencies alternately into the headphone by means of the double-pole double-throw switch and instructed the observer to vary the loudness of the 2048 cycle tone generated by audiometer *B* by means of the attenuation dial (calibrated in 5 db steps or intervals) until the two tones seemed equally loud. The observer was given as many alternate presentations of the two paired tones as he desired and was also permitted to manipulate the double-pole double-throw switch himself if he wished. After recording the intensity levels above the threshold values at which the two tones were judged equally loud, audiometer *B* was then regarded as the fixed or standard instrument and was left adjusted at the frequency and intensity levels just recorded. Audiometer *A* was next adjusted to generate a frequency an octave lower than *B*, or 1024 cycles, and the observer again instructed to vary the attenuation dial of the audiometer generating the lower frequency as before until the new pair of tones seemed equally loud. This procedure of loudness level matching was continued over the desired frequency and intensity levels generated by the audiometers,

the attenuation dial settings being recorded on the audiogram chart along with the threshold of audibility level.*

Comparisons of the data presented in Figs. 1 and 2 from normal ears show that the results agree reasonably closely over the frequency range from 1000 to 4000 cycles, thereby demonstrating the validity of the method employed over this frequency range in the present paper. The disagreements between the data shown in these figures are explained principally by the fact that the attenuation network of the Western Electric No. 2-A audiometer is calibrated in fixed intervals or steps of 5 db, instead of smaller intervals or continuous variability.

Fig. 3 shows a series of audiograms and graphs of equal loudness level contours from six ears of six observers. (Each individual in this group of curves presented bilateral thresholds of audibility which were nearly identical.) The heavy straight line at 0 level indicates the average or normal threshold of audibility. The nearest adjacent curved line indicates the threshold of audibility of the ear under experiment. The curves above the threshold curve are equal loudness level contours. Graph A shows that the threshold of this ear was nearly normal up to and including 2048 cycles, but was raised 70 db at 4096 cycles. Inspection of the equal loudness level contours of this ear indicate that the loudness resulting from increasing the intensity at 4096 cycles to 5 db above threshold, where there is a 70 db hearing loss, is equal in loudness to a frequency of 2048 cycles 35 db above threshold, where the acuity is approximately normal. The difference between the response of this ear and the normal ear is apparent by reference to Figs. 1 and 2, which shows that in the normal ear a frequency of 4096 cycles 5 db above threshold is equally loud as a frequency of 2048 cycles also 5 db above the threshold.

Graph B shows similar data from an ear with normal hearing up to 1024 cycles but with a raised threshold above this frequency level. No ear has been tested with normal acuity up to 512 cycles with a raised threshold above this level. Graph F, however, shows the threshold curve and equal loudness level contours of an ear with a slight hearing loss up to 256 cycles, with a considerably greater loss for higher frequencies. The curves of this graph do not show the marked deviation from the threshold at the low frequency end char-

*It is possible to verify the findings in the present paper with only one audiometer by alternating the frequency and intensity levels simultaneously until the observer judges them to be equally loud. Two audiometers permit more rapid alternate presentation of stimuli with less opportunity for error.

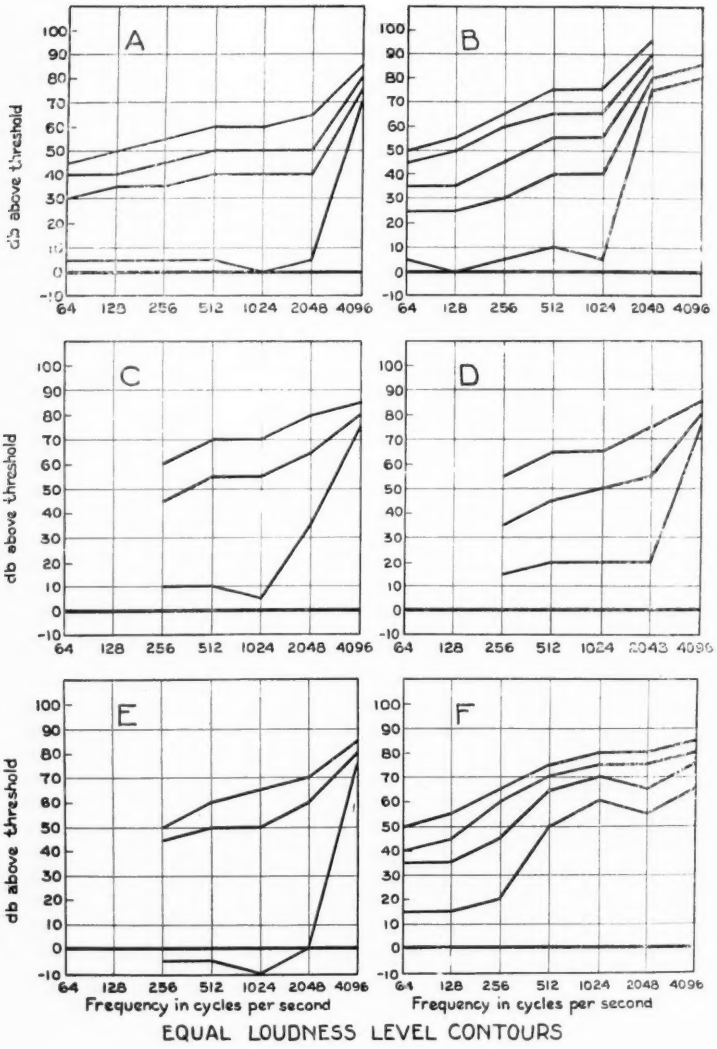
A
B
C
D
E
F
db above threshold
100
90
80
70
60
50
40
30
20
10
0
-10
64
128
256
512
1024
2048
4096
Frequency in cycles per second
EQUAL LOUDNESS LEVEL CONTOURS

Fig. 3

acteristic of the others, but show sufficient magnitude of divergence to be significant, especially when it is noted that Fig. 1 shows that the equal loudness level contours become closer and closer together, the lower the frequency, in the normal ear.

To date, equal loudness level curves have been obtained from twenty-one individuals with the type of hearing illustrated by the threshold curves of Fig. 3. Negative results were obtained from only one case. It was thought best to show representative curves at this time rather than statistical averages because too few individuals possessed identical thresholds over their audible frequency ranges to permit reliable accurate statistical analysis.

Note that the equal loudness level contours over the frequency range, throughout which the ears of Fig. 3 had normal thresholds, agree closely with the comparable data from normal ears shown in Fig. 2.

The curves shown in Fig. 3, plus additional data obtained but not shown, reveal two general tendencies worthy of note. First, the more pronounced the hearing loss the louder the auditory sensation when the intensity is increased a given number of db above the threshold. Second, the magnitude of the auditory sensation seems to increase more rapidly at intensity levels immediately above the threshold. This means that an increase in intensity of 5 db of a sound which is 2 db above threshold would sound relatively louder than a 5 db increase in intensity of a sound which is, e. g., 20 db above threshold. This latter conclusion is tentative, pending further investigation with attenuation networks calibrated in smaller intervals than the 5 db steps of the audiometer networks.

The finding that slight additional increases in intensity above the threshold of at least some types of pathologic ear result in much greater loudness increases than occur under similar conditions in the normal ear, makes possible a more reasonable explanation of several phenomena in relation to the hard-of-hearing ear. An additional explanation or interpretation of paracusis Willisii is made available. Let us assume an individual with a 60 db hearing loss for all frequencies within the most necessary range for intelligibility of speech sounds in a noisy environment which he just barely hears. In this environment individuals with normal hearing raise the intensity of their voices above the masking noise level in order to hear themselves with their normal speech loudness level and to communicate with their associates. If the general speech level is raised 10 db above the noise level, the loudness level of the speech will be somewhat greater

to the hard-of-hearing ear than the normal ear, if the conclusions by Guttman and Ham[4] and this study are correct. This aspect or viewpoint in regard to the explanation of paracusis Willisii makes plausible the unverified opinion that some individuals with the type of hearing loss just assumed can actually hear speech better in a noisy place than can a normal individual in the same environment.

This interpretation of the subjective response of the hard-of-hearing ear to intensity increments above threshold also throws considerable light upon the reactions of the hard-of-hearing to artificial hearing aids. It is a regrettable fact that many hard-of-hearing individuals refuse to wear artificial hearing aids because of an intense dislike of the microphone hiss or noise audible in the air-conduction type of receiver. Probably the reason is to be found in the explanation that variations in intensity of this noise above the threshold of the hard-of-hearing ear produce relatively much greater loudness changes than occur in the normal ear with a resulting greater interference with perception of the speech sounds. Most of the exceedingly small air-conduction receivers which are inserted into the external canal are also characterized by sharp resonant peaks which introduce considerable amplitude distortion in the reproduced speech. A given peak in an amplification system may create more detrimental amplitude distortion in the hard-of-hearing ear than in the normal ear because of the "magnified" subjective response of the hard-of-hearing ear to sound levels above the raised threshold. This loudness response of the hard-of-hearing ear must also be considered in the design of so-called compensated amplifiers for use by an ear with a greater hearing loss over a part of the speech frequency range than the remainder. If the thesis developed in the present paper be correct, the distortion of such a compensated amplifier would be greatest at the frequency levels where the hearing loss is most pronounced, because a frequency 5 db above threshold at that level would be experienced relatively louder than a frequency 5 db above threshold where the hearing loss is slight.

It is intended that the present paper be considered primarily as an introduction to a subject which seems worthy of further investigation. The following findings are tentative, pending the outcome of additional experimentation:

1. Slight additional increases in intensity above the threshold of the hard-of-hearing ear result in much greater loudness increases than occur under similar conditions in the normal ear. Or a given frequency a given number of db above the threshold of the hard-of-hearing ear results in a louder sensation of sound in that ear than

does the same frequency the same number of db above the threshold of the normal ear.

2. The greater the hearing loss the louder the auditory sensation when the intensity is increased a given number of db above the threshold.

3. The magnitude of the auditory sensation apparently increases more rapidly at intensity levels just above the threshold.

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LXXXIII

OSTEOMYELITIS OF THE SKULL ORIGINATING IN THE ETHMOID AREA

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and
Howard L. Norris, M.D.

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McKenzie of London, in 1913, first reported a series of cases of osteomyelitis of the skull, and since that time Kernan, Bourger and more recently Mosher, Kornblum and others have written excellent articles on this subject. None, however, so far as we have been able to ascertain, has reported the disease as originating entirely and exclusively from necrosis in the ethmoid area.

Most authors, especially Kornblum, have mentioned as causative factors and in order of frequency, paranasal sinus infection, mastoiditis, trauma, metastatic or hematogenous spread and syphilis. It is agreed by all that the frontal sinuses and mastoid region are by far the most frequent sources from which the disease may originate.

Early reports of osteomyelitis of the skull, doubtless due to defective x-ray technic, were superficial, no classification then being made except types, spontaneous and postoperative. All were presumed to be fatal. McKenzie mentions a mortality rate of 100 per cent, while later reports show there is great variation, not only in virulence but in extent of involvement of the bones.

Recent investigators have demonstrated that clinically there is a localized and a spreading type of the disease, and that the latter always is fatal, while the former is not necessarily so. The dividing line may be accounted for, at least partially, by variations in bone formation and also in type or grade of infection present.

The offending micro-organism in osteomyelitis of any type is, in general, staphylococcus, albus or aureus, and as a sequel of involvement of the skull bones, sequestrum, extradural abscess, meningitis, or thrombosis of the cerebral veins may be anticipated; while in the advanced stage of the disease marked changes in the blood picture usually are present. There is severe cranial pain and especially great sensitiveness of the skull on deep pressure.

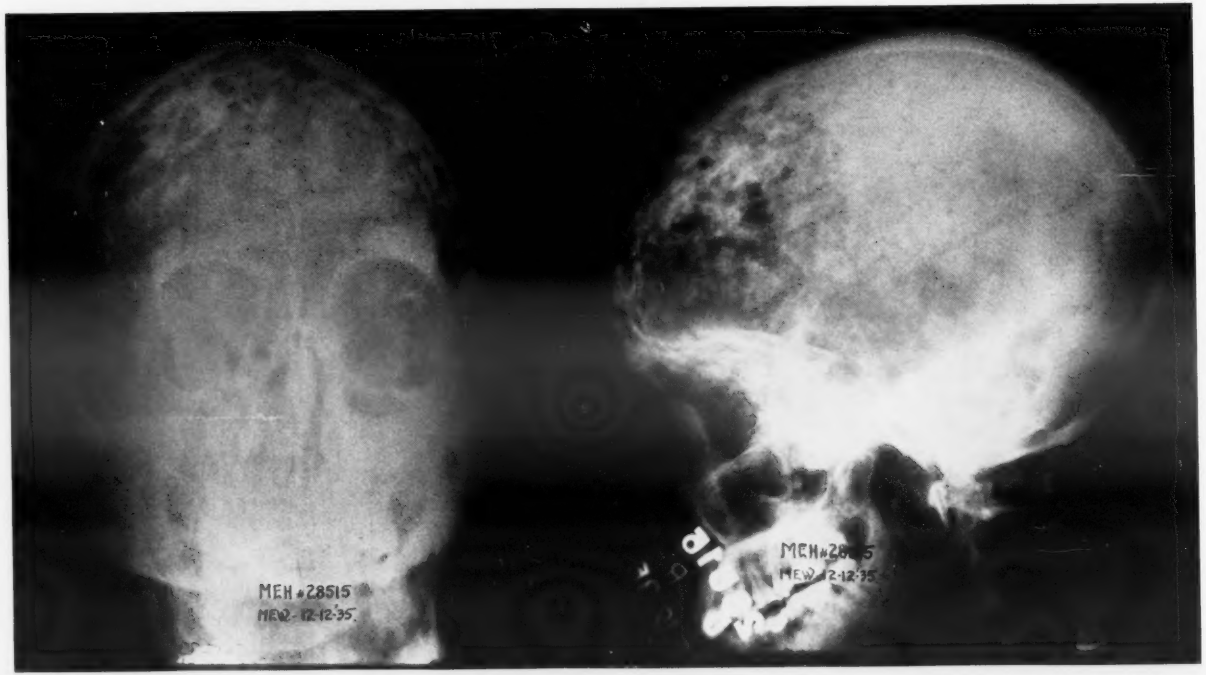

Fig. 1. Osteomyelitis of the frontal bone following an acute exacerbation of chronic ethmoiditis. Note the spreading type of the disease and involvement of entire frontal region. Lateral and anteroposterior views give a clear demonstration of extent of infection and bone destruction.

The disease is essentially chronic in its course, with progressive loss of weight and low grade temperature throughout. The latter stages are especially characterized by involvement of the cranial nerves, blood and lymph supply of and adjacent to the focus of infection.

The following case report covers a period of five or more months and embodies brief notes from beginning to termination. The x-ray and laboratory findings are included, as is the report from the post-mortem room.

REPORT OF A CASE

The patient, a female, aged 34, was first seen in consultation with Dr. R. J. Peters of Indianapolis on August 30, 1935. At that time the following history and clinical picture were presented:

Each orbital cavity was intensely edematous and very painful, especially on pressure; a profuse seropurulent nasal discharge was present. Further examination on the date mentioned showed a rather large amount of soft granulation tissue in the middle and superior meatal regions. The nasal septum was in the median line.

The history disclosed the fact that a purulent nasal condition had been present for a number of years, and also that some weeks previously surgery had been performed on the ethmoid labyrinth.

On September 10th the patient was referred to the Methodist Hospital for observation and further intranasal surgery. The x-ray findings at that time were as follows:

The frontal sinuses, rudimentary and very little evidence of infection. The ethmoid area on each side, very dense anteriorly and posteriorly, with suggestion of polypoid degeneration. The sphenoids completely filled with the products of infection. The sella turcica normal in size. No abnormal changes in the skull bones were demonstrated on above date.

The laboratory report on September 10th was as follows: Hemoglobin, 62 per cent; R. B. C., 3,160,000; W. B. C., 11,000; polynuclears, 93.

Further laboratory findings were more or less normal; urine was normal.

An operation was performed on the ethmoid labyrinth on September 10th and exenteration of all cells was attempted. Following this surgery the orbital cellulitis completely cleared up and the patient left the hospital seven days later with chart notation of condition, "Fair," and "Discharged at request of patient and family."

The patient re-entered the Methodist Hospital on September 20th for severe cranial pain and persistent septic temperature. This condition continued thereafter throughout the course of the disease, varying from time to time.

The nasal discharge and orbital cellulitis at this time were each negligible. The blood picture on date mentioned was entirely changed from the normal and progressed as such during the course of the disease.

The temperature at no time was above 100 degrees. The pulse was rapid and of poor volume but obviously not of the cerebral compression type.

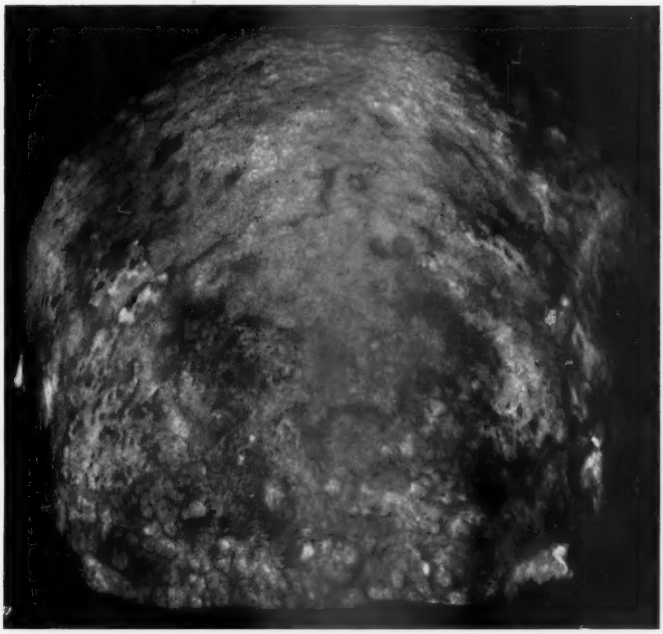

Fig. 2. The cranial vertex shown above illustrates the enormous destruction of bone present at death.

On November 4th an x-ray of the skull was done by Dr. Shifflett of the Department of Radiology, Indianapolis Methodist Hospital, revealed an osteomyelitis of the frontal bone covering an area approximately one and one-half inches above the upper margin of the frontal sinuses and extending well down towards each temporal region.

This condition increased steadily and at intervals of three or four weeks was checked, showing definitely the spreading type of the disease.

An operation was performed in early November, the character of which was elevation of the scalp, after incision through the eyebrows. All possible surface sequestrum was removed, the frontal sinuses opened and inspected and found to be practically normal. The necrotic bone and secretion was sent to the laboratory for section and culture, with the following report:

"Osteomyelitis with sequestrum formation, nonspecific in type. Culture of secretion showed staphylococcus aureus. The blood Wassermann was negative and the blood culture was negative." A blood transfusion was done at this time, with no appreciable result.

The patient died February 12, 1936, and throughout the two months preceding death presented many difficult problems. Left facial paralysis, almost complete,

was observed about November 20th, and a bilateral mastoiditis was recognized about December 20th. Neurologic findings prior to this period were entirely normal. The patient, during the latter half of January and until death, was totally blind and deaf. These facts point to involvement of the seventh, the second and the eighth cranial nerves. Mastoid surgery was not done, as the patient's physical condition contraindicated this procedure.

The blood picture at this time showed: R. B. C., 2,910,000; W. B. C., 14,000; hemoglobin, 62 per cent.

POSTMORTEM

The postmortem revealed a large extradural abscess, evidently originating in the cranial floor and extending anteriorly. Both mastoids were purulent and the trabeculæ in each were absorbed. There was a large area of sequestrum in each ethmoid region perforating into the cranial cavity. The frontal sinuses were again opened and found to be clean and free from observable infection.

These findings indicate conclusively that the origin of the osteomyelitis was in the ethmoid labyrinth and that necrosis in this area definitely was the causative factor in production of the skull pathology.

It will be recalled that the osteomyelitis was in a well advanced stage before the mastoids became infected. Undoubtedly the mastoiditis was due to the necrosing osteomyelitis and was in no way responsible for the skull infection.

HUME-MANSUR BLDG.

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LXXXIV

THE MANAGEMENT OF LABYRINTHINE INFECTION*

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There are few symptoms which cause more anxiety to the physician than the sudden onset of nystagmus and vertigo during the course of an acute or chronic otitis media. Every otologist knows that the labyrinth communicates directly with the subarachnoid space and that invasion of the internal ear often means that the pearly gates are ajar and ready to receive the patient. The problem of preventing a fatal meningitis, of doing the right thing or, what is perhaps more important, not doing the wrong thing for a given case of labyrinthitis—is one which presents difficulties even for the most experienced, if we are to judge from the extensive literature and differences of opinion which exist upon the treatment of labyrinthine infection.

In this brief report it is not intended to undertake an exhaustive discussion on the management of the various forms of labyrinthitis or to advocate a list of rigid indications for different methods of treatment. My purpose is to enumerate certain observations and impressions gained in handling cases of otitis interna complicating suppuration of the middle ear, with the hope that a record of personal experiences in this field, even if relatively small, may be of some value in stimulating thought and discussion upon this important subject.

The classification of labyrinthitis which I follow is the now familiar one taught by Professor Ruttin, based upon the various stages of labyrinthine involvement, namely: (1) Circumscribed labyrinthitis, indicating those cases which present signs of labyrinthine irritation, particularly at intervals, with a positive fistula sign; (2) Paralabyrinthitis indicating the same degree and type of irritation with a negative fistula sign; (3) Serous labyrinthitis, a stage of prolonged irritation or incomplete paralysis of the internal ear, the cardinal feature being the presence of some degree of function, either

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auditory or vestibular; (4) Diffuse manifest suppurative labyrinitis, giving the signs of an acute destructive lesion with total loss of internal ear function; (5) Diffuse latent suppurative labyrinthitis to indicate a dead labyrinth after the acute symptoms have subsided. This clinical grouping does not always correspond to the pathologic states which might be inferred from the various names but has been found as satisfactory as any other from a practical standpoint.

My experiences with the various forms of labyrinthitis may briefly be stated as follows:

1. *Circumscribed Labyrinthitis.*—We have had two of these cases in our clinic, both in chronic ears, in which the usual custom of doing a radical mastoid operation was followed with excellent results. In addition there was one case which developed an acute diffuse manifest suppurative labyrinthitis following an attic irrigation. The subsequent history of this patient has been previously given, but it may be mentioned that he recovered after a number of complications in addition to the suppurative labyrinthitis, necessitating a labyrinthectomy, obliteration of the lateral sinus, jugular ligation and drainage of an extradural abscess apparently originating from the labyrinth.

2. *Paralabyrinthitis.*—I have had two cases in private practice, both in acute mastoiditis, the symptoms first manifesting themselves several days following the simple mastoid operation. The diagnosis in each instance was made on the complaint of vertigo on change of position and the presence of spontaneous nystagmus to the diseased side under the Frenzel glasses when the patient moved his head or rapidly changed position. There was a spontaneous cure in each case after two or three weeks.

3. *Diffuse Labyrinthitis.*—It is the diffuse forms of labyrinthitis in their acute form, i. e., the serous and the diffuse manifest suppurative variety which often furnish the most baffling problems in management. As an example the following case is cited:

REPORT OF A CASE

CASE 1.—A boy, aged 7, at the time of his first admission to the ear department, had a tonsillectomy two years previously, followed by an acute otitis media in his left ear, which continued to drain until August, 1933, when an acute exacerbation occurred. Six days after the onset of the exacerbation he developed a left-sided peripheral facial paralysis and was admitted to the hospital. I saw him two days later and noted a horizontal-rotatory nystagmus to the diseased side. There was a polypoid mass in the external canal, issuing from a central perforation, much pus and débris, and low grade fever, but the patient was fairly comfortable except for moderate dizziness. Hearing tests showed definite hearing on the involved side,

although much reduced, and a caloric test indicated that the nystagmus to the diseased ear could be halted and reversed for a few seconds by cold water. A diagnosis of serous labyrinthitis was made and it was decided to treat the patient conservatively. He was kept under observation for nearly five weeks, at the end of which period the facial paralysis and the serous labyrinthitis were still present, although the latter had somewhat abated, but there was definite clinical evidence of mastoiditis requiring surgical intervention. A simple mastoid operation was performed with a burr and rongeur, using every precaution to avoid concussion or unnecessary trauma. In spite of this the patient developed, three days later, a diffuse manifest suppurative labyrinthitis with reversal of the nystagmus, which now was to the healthy ear and complete loss of function in the internal ear. There was fever not exceeding 102, no headaches and the spinal fluid was normal. The patient was kept in bed for six weeks with no further complication. During the subsequent months the facial paralysis gradually disappeared, but the mastoid wound refused to close and discharged sequestra from time to time. Eight months after the first operation a radical mastoid was performed, and since that time, with the exception of an occasional slight discharge, the boy has remained well.

The above case illustrates some of the difficult problems faced in the management of diffuse labyrinthitis—difficulties which the successful issue in this instance does not settle at all. The diagnosis of serous labyrinthitis at the outset was clear. The condition arose in the very early stage of the exacerbation and did not progress, so that in treating it conservatively we followed what I believe would be the practice of most authorities on the subject. The simple mastoid operation was performed finally because of the clinical evidence of mastoiditis and not because of the labyrinth involvement. Would an earlier operation have cured the mastoid infection and prevented the transformation of the relatively nondangerous serous labyrinthitis into the highly dangerous suppurative form?

It is commonly recognized that a serous labyrinthitis may change, as in this instance, into a suppurative one following a mastoid operation and also commonly stated that the use of gouges and the hammering on the mastoid is an important factor in bringing this as well as other complications about. Is the occurrence of suppurative labyrinthitis following an operation, or of a meningitis, or, even as I have had occasion to observe, the rapid activation of a previously latent and symptomless brain abscess, due to the mechanical trauma or to the increased inflammation of the residual structures in the temporal bone which may temporarily follow operative interference?

I am well aware that part of our treatment in the case just cited would be criticised as incomplete in many quarters. On the Continent it is customary in many clinics to operate at once on cases of diffuse manifest suppurative labyrinthitis, performing a radical and labyrinth operation at the same sitting, as advocated by Professor

Ruttin. I have had occasion to see about a half dozen cases of acute diffuse suppurative labyrinthitis, all of whom recovered without, to my knowledge, any further complication from their acute illness, simply by rest in bed and conservative treatment. It must be emphasized, however, that all of these occurred during the course of a chronic ear infection.

What about a simple or a radical mastoid operation performed during a diffuse manifest suppurative labyrinthitis? This, to my mind, is a highly dangerous procedure, worse than not operating at all. This was illustrated to me in dramatic fashion several years ago in a case under treatment by a colleague whom I chanced to be visiting at the time. I was shown at the hospital a man who had two days previously, during the course of a chronic otitis media, developed nystagmus, vertigo and vomiting. At the time I saw him he still had nystagmus to the opposite side, total loss of hearing and no caloric reaction, but was quite comfortable and had very little fever. The otologist insisted upon doing a radical mastoid operation and removed a large cholesteatoma. The patient did well for three days and then developed a fatal meningitis, which was corroborated at autopsy. From my observation of the patient I have a strong impression that he would have recovered from the acute stage of his labyrinth infection if he had been left alone.

In the treatment of the chronic stage of labyrinth infection or the so-called latent diffuse labyrinthitis I was exposed, like other men during my Vienna days, to two diametrically opposite teachings. Professor Ruttin stated that such a case should not be operated upon except for the usual indications in a chronic running ear, but that if it was operated at all a combined radical and labyrinth operation should always be performed at one sitting; Professor Alexander taught that a careful radical during the quiescent stage was, barring other indications, sufficient. It has always appeared to me that in those cases where a dead labyrinth has existed for several months or years, and particularly where the patient cannot recall vertigo or nausea, i. e., where the so-called gradual symptomless death of the labyrinth probably occurred, the chances of an actual suppuration still being present in the labyrinth were not great and that the more conservative procedure would do just as well. I have had occasion to do a radical mastoid operation alone in four cases with long-standing dead labyrinths, and in none of these did a meningitis occur. In one case, however, the operation was followed by the lighting up of a chronic cerebellar abscess which had previously given no symptoms. The abscess was located and drained, but the patient could not be

saved; death was from encephalitis. I do not see how the performance of a labyrinth operation simultaneously with the radical mastoid operation would have averted this occurrence.

In summing up our experiences, one may perhaps be permitted a few general reflections on the subject of the treatment of diffuse suppurative labyrinthitis. The theoretical basis for this, as laid down by the Neumann clinic, is simple: in the manifest or acute variety the infection is in danger of spreading to the meninges, therefore the labyrinth should be drained; in the chronic or latent form, if a mastoid operation is indicated, the labyrinth is to be looked upon as another infected cell which must be opened to avoid a possible exacerbation. In practice, however, matters are much more complicated. The labyrinth operation, while not a formidable procedure ordinarily, can be quite difficult where Trautman's triangle is narrow and carries a danger to the facial nerve far greater than in the usual radical mastoid operation. One would perhaps ignore these drawbacks were we certain that drainage of an acute suppurative labyrinthitis would always prevent the meningitis which is so feared. Actually the results of Lund and other conservative otologists compare quite favorably with those of the more radical school. In commenting upon the management of chronic or latent suppurative labyrinthitis, it is to be noted that Turner and Fraser, as a result of their large experience, take the same point of view as in our clinic, namely, that such a case requires only a radical operation. I have reserved the labyrinth operation for those cases in which an increasing spinal fluid cell count indicates that a meningitis is threatened or has occurred as a result of a manifest or latent diffuse suppurative labyrinthitis and in those instances where for other reasons it is necessary to open the mastoid during or shortly after an acute suppurative process in the internal ear, such as the young man whose case was mentioned under "circumscribed labyrinthitis"; the technic preferred is that of Neumann as described by Schlander. There may be other instances occasionally where labyrinthectomy is indicated, such as where a cholesteatoma is seen to invade the internal ear or sequestration is found at operation, but the above are the only definite reasons followed in this clinic.

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LXXXV

OPERATIVE AND NONOPERATIVE TREATMENT OF ETHMOIDITIS*

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The purpose of this paper is to speak in a brief way of the inflammation of the accessory nasal sinuses which is designated as "ethmoiditis," and to mention some of the methods, both operative and nonoperative, which we have at our disposal for the relief of this symptom complex and its complications and to attempt to decide upon their relative merits.

As one attempts to investigate the development of this subject he is overwhelmed with a mass of literature written upon the ethmoid and is made cognizant of the fact that for the past fifty years otolaryngologists have found this subject an interesting and puzzling one, and that while there has been some progress made in the development of the nonoperative treatment, there is still much to be desired. Jonathan Wright once said that he hoped or believed that the time would come when there would be no operative work done on the accessory sinuses, and I sincerely hope that his prophecy may be realized, but with our present understanding and treatment of this condition I feel that the millennium is still far away. Hence I do not feel that I should be apologetic for this paper, but rather that I am offering for discussion a subject which is of common interest and will be so as long as we have rhinologists and the rhinologists have patients. However, within the past fifteen or twenty years, we have had the advent of some excellent investigators who have given of themselves unsparingly that we might better understand the anatomy of the ethmoid and have devised for us some excellent operations, both intranasal and external, for the relief of this condition when it becomes operative.

Two of the many things which have interested me in the study of this disease have been:

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1. The difference between acute catarrhal and acute purulent ethmoiditis. I know that Skillern, in making this differentiation, speaks of the acute rhinitis or common cold as being an example of the acute catarrhal type and that the concomitant sinusitis of measles, scarlet fever and the other exanthemata are an example of the acute purulent type. However, with most profound respect and due deference to such a great teacher and authority as Skillern, I would like to venture to say that this part of the classification was existent before the development of the bacteriology of the sinuses and that it is cutting corners very sharply to make such a differentiation at this time. I am inclined to believe that the acute catarrhal type is a holdover from the old classification when the rhinologists dealt with an allergic condition and were not familiar with its etiology.

2. The other is, why we speak of ethmoiditis so often when in reality we are talking of pansinusitis? Is it a habit we have gotten into or perhaps it is because of the fact that as the ethmoid is the keystone of the whole sinus group on either side, the term "ethmoiditis" is an easy way of saying "pansinusitis." We speak of ethmoiditis in children and, while we give them constitutional, nonoperative treatment, we nevertheless pay great attention to the proper irrigation of their antra, if we are to hope for success. Also we very seldom see a sphenoiditis or a frontal sinusitis which is not secondary to ethmoiditis. We know that the latent antrum prevents the resolution of acute ethmoiditis, and an infected antrum of tooth origin will very quickly cause an inflammation of the ethmoid and, if untreated, very readily develops into a hyperplastic purulent ethmoiditis or pansinusitis on the affected side. Hence, it seems reasonable to suppose that we have been using the term "ethmoiditis" rather loosely; whether we change or not remains for the more learned and wiser members of this society to determine.

Therefore, in making our classification of this condition at this time, we will speak of ethmoiditis as being acute purulent, chronic hyperplastic and hypertrophic, and chronic purulent hyperplastic and hypertrophic, and if we may add, for the purpose of discussion, vasomotor rhinitis and ozena.

I mention ozena and allergic sinusitis because the ethmoid is definitely and wholly involved in these conditions and for the fact that in the clinic of Jones at the Manhattan Eye, Ear and Throat Hospital, we have recognized in the past decade a marked decrease in the number of patients suffering from ozena and a marked increase in those suffering from allergic sinusitis.

The decrease in ozena we feel cannot be contributed to the demise of the bacillus of Perez or to a decrease in immigration, but rather to the improvement of a hygienic condition throughout this nation among the less properous families, the advance in pediatrics and the investigations and investigators in biologic chemistry who have so wonderfully collaborated with the clinicians and given us our present knowledge of the value of balanced diets, vitamins and the necessity of sunshine and fresh air in combating local manifestations of metabolic disturbances.

In allergic sinusitis, the precursor of one of our most difficult problems in treating ethmoiditis, we find a somewhat more perplexing problem for solution. Here we must deal with exhaustion neuroses, endocrin imbalances and the general anaphylactic sensitivity of the somatic cells as a whole to organic or inorganic toxins, or to the development within the body complex through metabolic disturbances, due to chemical imbalances, of other special sensitivities for foreign proteins which may be expressed by local manifestations in the sinuses of the condition which is variously described by such terms as vasomotor, allergic ethmoiditis or sinusitis. There is little doubt that the allergists have worked very hard to understand this problem better and that they have in many cases been able to help their patients through desensitization, but their best and most complete work has been accomplished in conjunction with the rhinologists, the biologic chemists and the dietitians. It is to be regretted that some of the pioneers in the field of allergy still feel that they are sufficient unto themselves. Let us hope that the next decade will give us more advances than we have had in the past decade.

Some rhinologists have claimed excellent results in treating this condition by means of ionization. The writer has not had any experience with this form of treatment and hence hesitates to pass judgment. However, he believes it is still very much in the experimental stage. We will venture to say that any method which treats effects and not causes, whether it be electrically, medically or surgically, is bound to be abandoned.

ACUTE ETHMOIDITIS

In speaking of the nonoperative treatment of this condition, it is not the writer's desire to bring before you for consideration all the various methods that have been advocated or to herald a new form of treatment, but rather to mention a few procedures which are and have been very popular among many rhinologists and to analyze their rationality. Are they indicated? Do they do more harm than

good? Should they be continued as a part of the treatment of this condition?

The first of these is dry suction. It is the writer's belief that actual harm is done by the employment of this procedure in acute ethmoiditis, by causing an undetermined amount of trauma through the dilatation of the mucous membranes of the nose and sinuses, thus injuring these tissues to the degree that they offer less resistance to the offending organism, interfere with the vasomotor control and cause an exaggeration of the symptoms present. Also, that by creating a partial vacuum in the sphenoid and frontal sinuses during the application of the suction, there is great danger of aspirating the infection deep into these areas. This is particularly possible and probable when the nose is not first cleaned of the purulent discharge so often confined to the area under the middle turbinate. In my early experience I was guilty of using suction as a part of my method of treatment, and feel that I have been responsible for some delayed resolutions and some acute frontal conditions which otherwise would not have occurred.

Another procedure which might be relegated to the same category is suction irrigation. Those of you who do use, or have used, this treatment in acute sinusitis have experienced the aggravated condition in which some of these patients return to the office twenty-four hours after being treated by this method. This aggravation may readily be explained by the fact that we very seldom see these cases of acute ethmoiditis until two or more days after the onset of the symptoms. In the majority of cases the infection at this time is confined mostly to the dependent sinuses of the nose, and there is usually a fair amount of purulent secretion under the middle turbinate. It is possible that the cells on one side or groups of cells on one side, or groups of cells on different sides may be in a greater stage of inflammation than other groups on the same side or both sides. By the use of suction irrigation, the irrigating solution is made the vehicle for the transfer of the infection to all the sinuses of the head, and the vacuum formed by the suction invites the infection into the sphenoid, the frontals, the deeper cells of the ethmoid, and the more distant parts of the antral mucous membrane. In other words, in the use of suction irrigation, we may readily change a mild infection of the sinuses to an overwhelming one which in some cases leads to an unnecessary loss of time to the patient and possibly complicates matters with an acute ear. I do not say that suction irrigation has not a place in the treatment of sinuses, but we believe it is contraindicated in the treatment of all acute sinusitis.

SURGICAL TREATMENT

There is very little to be said for the surgical treatment of acute ethmoiditis, except when we speak of the complications of acute ethmoiditis, and the most common complication, as you would expect, which may be considered from the surgical standpoint, has been antritis. This complication, we believe, is most often the one responsible for delayed resolution in these cases, and if an acute ethmoiditis continues over a period of more than two weeks, we feel justified in investigating the antra by washing through the natural opening or above the middle turbinate, and it has been our experience that a great number of them are immediately relieved and started on their way to normal resolution.

The next complication is the acute frontal and acute sphenoiditis. It is our belief that many of these cases will resolve themselves if we persist in palliative treatment long enough, relieving the patients by the use of heat, salicylates and analgesics. There was a time, not so many years ago, when an acute frontal lasting two or three days was sufficient excuse for some intranasal surgery, and while we perhaps helped some of these patients to be relieved of their acute symptoms, I believe we did more harm than good and often set up a chronic ethmoiditis which eventually went on to a purulent hyperplasia with its attendant exacerbations. It is now our practice to make surgical intervention the last act in treatment, even though it be necessary to hospitalize the patients for a period of a week or ten days, in carrying out our nonoperative treatment.

The most important complication of this particular phase of ethmoiditis is the subperiosteal abscess or orbital cellulitis and the management of the same, particularly in chilldren. This requires a fineness of surgical judgment which cannot be ovestimated and requires that the rhinologist call on both the laboratory and whatever other consultants he may have at his service, whenever there is doubt as to the proper time for surgical intervention. We see a great variation in the relative severity of this complication, from a slight edema of the eyelid, which subsides without any interference, to the extreme case with severe edema, proptosis and fixation of the eyeball, etc. Our procedure in this type of case has changed somewhat in the last decade. At one time we felt justified in proceeding surgically at the first sign of orbital involvement, and very often found what appeared to be a perfectly normal looking ethmoid without presence of pus. At present we watch these cases for a period of a week, ten days or two weeks, depending upon the condition of the patient's

temperature, the amount of swelling, the blood findings, etc., before interfering surgically. It is surprising how many cases will resolve themselves through the simple application of supportive treatment, local treatment to the mucous membranes of the nose, as hot saline irrigations, and the irrigation of the antrum of the affected side. I believe that as many cases of orbital edema in children are due to an extension from the antrum as from the ethmoid.

If it becomes necessary for surgical interference the usual external ethmoid incision is made and the tissues elevated along the floor of the frontal and the os-planum until the abscess is reached and evacuated. In children we feel that this is all that is necessary, and that in 80 per cent of the cases it is all that is indicated for a complete cure of the case. Unless we discover some gross pathology on the floor of the frontal or the os-planum of the ethmoid, nothing is done to these areas. However, if we do find granulations, we believe that nature is fighting a losing battle, that the perforation is due more to erosion and infection of the bone than to a dehiscence or rupture of the ethmofrontal suture, and we are justified in resorting to a radical procedure to establish drainage. This conservatism, if you care to call it such, has been a result of our observation of the association of early and radical operative procedures in these cases and the occurrence of osteomyelitis of the frontal bone. This is perhaps nothing new to some of you present, but nevertheless it is still a practice in some communities and we cannot emphasize too strongly the fact that many of these cases require only incision and drainage for their relief and cure.

In adults the story is different. We find in most cases of orbital infection that the infection is usually the result of frequent and severe exacerbations of a chronic hypertrophic purulent sinusitis with a definite perforation and extensive disease of the frontal and ethmoid sinuses, and very few of these can be cured without thorough exenteration of the ethmoid and removal of the floor of the frontal.

HYPERTROPHIC AND HYPERTROPHIC PURULENT ETHMOIDITIS

To speak fully of the nonoperative treatment of hypertrophic and hypertrophic purulent ethmoiditis, it would be necessary to run the whole gamut of remedies and methods of treatments which have been in vogue or have come into vogue during the past ten years. Such a procedure would be tiresome, is unnecessary and would avail nothing. We must, however, mention such a monumental work as the investigations and the methods developed by Proetz and also pay

tribute to the closer association of the internist and rhinologist. Proetz's work offers us a modus operandi for the diagnosis and non-surgical treatment of these cases we never had before; also a better understanding of the type of case which may respond to surgical intervention.

The use of vaccines in this condition has been rather disappointing, and when we are just about ready to give up all hope of aid from this source we learn of the development of a new type of vaccine, developed by Krueger, known as an undenatured bacterial antigen and used with most gratifying results by Kracow. The method of the preparation of this vaccine makes it a filtrate. It is composed of two components, first, a water-soluble fraction; second, a colloidal phase of bacterial fragments which can be demonstrated in the ultramicroscope. It is possible that our results may be much better with the use of this substance.

This type of ethmoiditis, which constitutes a large part of our rhinologic practice, gives us the most trouble and taxes our judgment to the extreme. It is responsible for more complications than any other type; the vascular and lymphatic changes which are present in the tissue and the direct association of the vascular systems of the ethmoid and orbit are responsible for much of the eye trouble. The conjunctiva, the cornea, the sclera, the optic nerve and the intraocular tissue are likely prey for the toxins for this area. Very often the rhinologist is appointed the court of last appeal, as to whether or not surgical intervention shall be instituted or advised in such cases as retrobulbar neuritis, chorioretinitis and other destructive lesions of the eye. In some cases he has no other alternative but to operate; in others, he will most likely arrive at his decision by a process of elimination with his own particular experience, knowledge and reasoning thrown into the balance.

With the decision to operate final, we prepare to do an intranasal ethmoid unless there is some contraindication to such a procedure. It may be that the oculist prefers the external operation, or there may be some superorbital dells which the operator does not think he can reach by the intranasal method. Perhaps, from his examination of the x-rays with the roentgenologist, he decides that there is too much sclerosis present to preclude a satisfactory ethmoidectomy by the intranasal route. If there is a thickening or a deviation of the septum, this should be removed first, and if a large bulbous middle turbinate exists on the opposite side, which might interfere with the view of the ethmoid on the affected side, this is removed before the ethmoidectomy is started.

Our operation consist of a modified Mosher-Faulkner procedure, inasmuch as we use some of the instruments and the technic of each of these methods of operating. We find it more convenient and we think it is safer to enter the aga-nasi cell with the Mosher curette and work our way back to the pterygoid plate and into the sphenoid with this instrument, and then with a curved Strychland we cut through the middle turbinate at about the junction of the posterior and the middle third. Then with a pair of Coffin forceps, introduced under the middle turbinate and as well into the anterior cells, as much of the middle turbinate and the anterior cells as possible are removed in one bite of the forceps. This opens the ethmoid labyrinth and we proceed with the extirpation of the remaining tissue by the use of the Coffin forcep, the Faulkner ring and spade curettes. The sphenoid opening is enlarged, depending upon the consistency of the anterior wall, with either the Mosher or the sphenoid punch. We do not advise or use biting instruments unless such is absolutely necessary to give us a clean looking field. Care is taken to exenterate the ethmofrontal cells and introduce a Goode rasp without force to determine the size of our frontal opening. This in brief is the procedure we use, and it may be varied from time to time as our judgment dictates. Whenever we do an intranasal ethmoid, it has been our practice to also do an intranasal antrum on the same side for the obvious reason that many of the antra will become secondarily infected in the process of repair of the ethmoid area. We feel this is a wise thing to do.

CHRONIC HYPERPLASTIC AND CHRONIC HYPERPLASTIC PURULENT ETHMOIDITIS

Considering the known pathology of this type of ethmoiditis and the excellent work of Eggston on the subject, "Pathology of Chronic Sinusitis," we feel there is very little to be said for the nonoperative phase of treatment. I realize that some writers have claimed excellent results through the medium of allergy, the proper desensitization, food eliminations, endocrin balances, etc., but it has been our misfortune not to have had much success in this condition from these methods of treatment, nor has it been our good fortune to have seen any of the good results. We know that radium has been used in several different experimentations during the past ten years, but has been discarded as having little or no value in those cases which are complicated with asthma, or bronchiectasis. We have failed to see any form of nonoperative treatment render any material benefit to many patients suffering from well established bronchiectatic or asthmatic symptoms, hence we have turned to the operative treatment for results.

symptoms, hence we have turned to the operative treatment for results.

In speaking of the operative treatment, it is necessary that we establish the fact that we do not operate on any cases of asthma or bronchiectasis unless they have been thoroughly gone over from the allergic point of view and it is well established that our known non-operative methods of treatment in this field can avail the patient little or nothing. Only then do we feel that we are warranted in starting with our most radical operative treatment.

The amount of operating done varies with the history, clinical and x-ray findings. Given a case of mild chronic bronchiectasis with a protracted cough, but not a large volume of bronchial secretion, who shows on clinical examination a definite hyperplastic ethmoiditis without any secondary infection, and the x-ray plates give evidence more of an osteoperous than areas of osteosclerosis. Such a case, in our opinion, would do well with a double intranasal ethmoidectomy with an enlargement of the sphenoid osti and a double intranasal antrum. In the extreme hyperplastic cases, with considerable polypoid degeneration and suppuration, with definite areas of sclerosis, is the type of case for which we would advise the external ethmoid route. Whether the case be one of bronchiectasis or asthma, we usually divide the operative treatment into two parts.

1. A double external ethmoid operation is done after the method of Lynch or Smith, endeavoring to be as thorough as possible in removing all infected material from the frontals, ethmoid and sphenoid. This first operation is done under a general anesthetic.

2. The second operation, which may follow ten days or two weeks after the first operation, is usually done under local anesthesia. At this time a Caldwell-Luc operation is done on both antra, being careful to remove all the polypoid membrane from the antrum, taking down the naso-antral wall and making a thorough inspection of the inferior turbinate to determine how much disease is present in this tissue. If it is boggy and polypoid, the whole turbinate is removed at this time. We feel that in the previous cases, in which the inferior turbinate was not disturbed, it very often interfered with the proper drainage. A month or two after operation we often found two large polypoid inferior turbinates which interfered with the resolution of the area operated upon and the improvement of the patient. Hence we now feel thoroughly justified in removing these turbinates and have as yet to notice any untoward symptoms in the patient even after a period of five years.

The removal of the inferior turbinates has been criticised, but we felt that inasmuch as the purpose of the operation is to remove all infected tissue, there was just as much pathology in these inferior turbinates as there was in the sphenoid or the frontal sinuses.

There has been considerable difference of opinion as to the value of the operative method for the cure and improvement of asthmatic patients. The literature is full of conflicting opinions; nevertheless, we feel our results have been satisfying enough to both the patients and the operator to continue our work. We are certain that all of these patients have been helped and some have been cured up to the period of eight years. If we can cure three out of ten, help five (60 per cent), and improve two (40 per cent), then our work has not been in vain.

And so we bring the paper to a close with the conclusion that while there has not been a great improvement in the nonoperative treatment of ethmoiditis in the last decade or two, much time, thought and energy are being expended in that direction at present.

The operative technic and type of operation have vastly improved, thanks to such men as Skillern, Mosher, Ballinger and Hajek. However, our goal is not the operative treatment but to prevent our patients from getting in such condition that they need an operation.

140 EAST 54TH ST.

LXXXVI

HYPOTHYROIDISM IN THE PRACTICE OF OTORHINOLARYNGOLOGY*

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CINCINNATI.

Despite the fact that the otolaryngologist's horizon is constantly broadening and that daily attention is being given to various diseases of infancy and childhood and to many systemic conditions in the adult, all of which have a more or less direct bearing on pathologic conditions of the ear, nose and throat, comparatively little notice has been taken of the rôle played in diseases of these same organs by the glands of internal secretion. Of the various endocrine dyscrasias, those of the thyroid gland are of particular importance in our field. It is amazing to find the number of these patients who have been under treatment for years, and often in competent hands, without this fundamental cause ever having been discovered.

Although the theory of internal secretion as such is commonly considered to rest primarily on the work of Claude Bernard and Brown-Sequard, it is not to be supposed that ductless glands were unknown before their pronouncements were made. Burdach, Mueller, Henle, Koelliker and Bordeu all recognized and described ductless glands, but only on the basis of their anatomic appearance and location, and presumed that their function was to bring about some change in the composition or elements of the blood passing through them. Although Vulpian, de Cyon, von Schroeder, Schiff, J. S. Riverdin, A. Riverdin, Kocher, Gull and Ard reported clinical observations, including among other things the production of a myxedematous state following thyroidectomy, the theory of internal secretion remained a theory, substantiated only by these clinical observations and occasional therapeutic proofs, until Brown-Sequard, by intensive work on the subject, between the years 1889 and 1891, showed that "these specific soluble products pass into the blood, and, by its agency, react upon the cells of other atomic elements of the organism."

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In 1905, Starling named these "specific soluble products" hormones. In 1890 and 1891, Vassale in Italy and Gley in France showed that experimental animals whose thyroid glands had been removed could be kept alive by the injection of thyroid extract.

It was quickly found that thyroid extract has the property of augmenting respiratory and nitrogenous changes, the reverse of which obtains in thyroidectomized and myxedematous subjects.

For a proper understanding of the profound influence of thyroid, it is, of course, necessary to have a clear picture of the physiology of the gland. Some details of its physiology are still clothed in obscurity, particularly those having to do with the inter-relation of the various glands of internal secretion, but certain points of thyroid physiology have been proven.

The thyroid gland produces an internal secretion, or hormone, which is essential to the normal growth and metabolism of the individual, and which has a pronounced action on the nervous functions. It governs the building up of cells and, therefore, the formation and growth of tissues, and it further regulates the destruction of the albumin molecule and governs the process of elimination of waste materials. And, finally, it plays a definite rôle in the resistance to infections by governing the production of alexines and opsonins, which, although formed by the liver, are apparently controlled by the thyroid gland inasmuch as its under-activity decreases the secretion of the necessary exciting hormone. The last named physiologic action was at first stated empirically and only partially correctly, but the work of Sajous, Fassin, Stepanoff, Marbé, Chittenden, Justus and others has shown that the production of alexines is augmented by the stimulating effect of the thyroid hormone on the activity of the adrenals and, through these organs, on general oxidation and metabolism, while the opsonic factor is probably actually an increased vulnerability of bacteria, their toxins, endotoxins and toxic wastes to the digestive action of the phagocytic or cellular defensive ferments on the basis of phosphoric acid metabolism, which is greatly influenced by the thyroid gland because of its iodine content. The reduction of fat in obese subjects probably depends on this same phosphoric metabolism as well as upon the release of stored water.

The faulty elimination resulting from an underactivity of the thyroid causes infiltration. This infiltration is represented grossly by puffiness of the face and generalized edema which does not pit upon pressure. The infiltration which takes place in other systems and tissues produces symptoms dependent upon the function of the

involved structures. For example, constipation, so frequently met with, is due to atony of the intestinal musculature and impaired function of the intestinal glands. The rheumatoid pains, which are also frequently encountered, are due to muscular, ligamentary and articular infiltrations. Then there is the group of symptoms dependent upon epidermal involvement, such as dry desquamation of the skin, changes in the finger nails, and alterations in and loss of hair, particularly of the outer third of the eyebrows and the temple regions of the scalp. Finally, there is the general set of subjective symptoms due to chronic constipation, generalized infiltration and auto-infection, namely, chronic fatigue, drowsiness and generalized indefinite aches and pains.

When all of these changes are present in a sufficiently marked degree to produce the picture of true myxedema or cretinism, the diagnosis is obvious at a glance. Our present discussion, however, is of benign myxedema, or, as Hertoghe termed it, the "forme fruste." When all the signs of this benign type are evident, it is termed the "Hertoghe syndrome." A patient presenting all of the evidences of this condition might be described as follows: He looks and feels older than his years. He awakens tired after a long night's rest and is loath to arise in the morning. He has a comparatively expressionless face or looks constantly surprised, due to the thinning of the outer third of the eyebrows. His hair is prematurely gray, fine in texture and receding at the forehead. He is habitually constipated and often nauseated. He has frequent headache, either frontal in type, radiating behind and above the orbit, or of a migraine character. He has almost constant backache and rheumatoid pains in various joints. His feet and heels ache, as does the back of his neck. His eyes are puffy and he has unevenly distributed fat. He is apt to be sad, depressed and worried most of the time, and finds it difficult to concentrate, and when he has thought out a subject the effort required to express himself is too great. His memory is faulty. He has genito-urinary symptoms in the form of frequent micturition due to inflammation from the desquamated cells of the bladder mucosa. His nose is stuffy, his hearing is impaired and he has tinnitus, sometimes accompanied by hallucinations of hearing. He has frequent attacks of dizziness. There are also visual hallucinations and there is imbalance of the muscles of the eye. His voice is husky and he has frequent colds. He is subject to all manner of superficial skin infections. His skin is dry and scaly, particularly over the elbows, knees and extensor surfaces. He is always chilly, this sensation being aggravated by the slightest draft, and his hands and feet are habitually cold. When his temperature is taken it is found to be subnormal, particularly in the

late afternoon. He has cardiac dyspnea which is frequently confused with asthma. He has no interest in what goes on about him, he has no appetite, and he drags himself unwillingly from one endeavor to another. In many cases other members of his family, antecedent, descendant or collateral bear the same stigmata.

I do not mean to infer, of course, that every subthyroid individual presents this composite picture, but if even a few of these general symptoms occur a basal metabolic determination should be made, for a little thought will indicate that all of these depend upon infiltration of the organs in question.

Occasionally one finds symptoms of hypothyroidism and hyperthyroidism in the same patient, the latter undoubtedly being a reaction to the former, which is always of long standing, and in these cases, diagnosis having been made, it is important that the physician have courage enough to prescribe the necessary medication in spite of a possible temporary flare-up of the hyperthyroid symptoms.

In a recent series of case histories of sixty-five of our subthyroid patients which I have analyzed, eighteen were male and forty-seven female. This would seem to bear out the contention of many writers that there is a preponderance of subthyroidism in females due to the extra demand on the thyroid gland during the menstrual cycle, largely, it is thought, in order to aid in increasing the plasticity of the blood.

Certain definite etiologic factors producing a state of thyroidism are recognized, such as complete or nearly complete thyroidectomies, and the damage sometimes consequent to radiation therapy of the thyroid gland. Curschmann observed that hypothyroidism was increased in post-war days, stating that the universal malnutrition resulted in a decrease in the main building stone of thyroxin, especially tryptophan. Other etiologic factors which must always be considered are illness and worry, together with unusual mental strain. It is reasonable to suppose, therefore, that the strain of life as it is lived today may be the answer in part to the increasing number of hypothyroid individuals, together with the fact that physicians are gradually becoming more alert in recognizing the condition.

The laboratory confirmatory tests to substantiate the clinical diagnosis which are most freqeuntly used are, of course, the dextrose galactose tolerance test and the determination of the basal metabolic rate. In this connection it is well to remember that an error in the basal metabolic test can be made only on the positive side, and it is therefore not necessary to have a figure below the arbitrarily set

—10 of the normal limit range. In fact, I am convinced that many patients who need thyroid medication and who cannot improve without it are deprived of this improvement because the basal metabolic test resulted in a —4 or —6 reading. Kimball reports an interesting case in which the clinical diagnosis seemed certain, but the basal metabolic test resulted in a rate of +60. This was checked with the same result, and only then was the patient checked and found to have a large perforation in the tympanic membrane. When the auditory canal was tightly closed and the test repeated, the basal metabolic rate was —20.

The differential diagnosis is commonly between neurasthenia, some of the anemias, tuberculosis, various psychoses and, of course, disturbances of other endocrine glands.

When a child has repeated colds without some discernible cause, or despite the removal of such a cause as infected tonsils and adenoid, inquiry should be made as to its mental development and capacity for learning, and also regarding nervousness and irritability. Kimball has reported on the study of mentally retarded children in the schools of Detroit and Cleveland, in which he found that the only cases in which the mental defect was traceable to some glandular deficiency were those of congenital hypothyroidism. Comparatively few are cretins and therefore recognized in early childhood, consequently the hypothyroidism is apt to go unrecognized and the child to be blamed for lack of concentration, subnormal intelligence and mental retardation. Kimball points out that one of the simplest indications of subthyroidism are structural defects of the capillaries of the nail grooves.

The age range in our cases was between six and fifty-five, the majority, however, being in the second, third and fourth decades of life. Twenty-three had symptoms referable to the ear, eighteen to the nose and thirteen to the throat, eleven having miscellaneous symptoms, such as headache, trigeminal pain and choking spells.

The external auditory meatus suffers in common with the epidermis elsewhere, and we have found many cases presenting eczema of the canals with scaling, redness and cracking, which have resisted all forms of local treatment.

One of our commonest occurrences has been that of repeated furuncles of the external canal in patients with normal blood sugar and no other demonstrable cause, originating in the subthyroid state.

The deafness frequently associated with tinnitus and sometimes

with attacks of vertigo is considered by Drury, W. S. Bryant, Moehlig, Smith and others as being due to any of or a combination of the following: swelling and infiltration of the orifice of the eustachian tube in connection with the same condition of the nasal membranes; infiltration of the nerve centers; relaxation of the temporomandibular joint by the stretching of the ligamentous structures, affecting secondarily the muscles surrounding the eustachian tube, this relaxation being similar to that frequently seen in the plantar region and elsewhere, and a possible tetanic muscular contraction of the intratympanic muscles resulting from derangements of the pituitary as well as of the thyroid gland.

A problem occasionally confronting every otolaryngologist is that of nasal obstruction, watery discharge and repeated colds, which persist after apparently well indicated operations have been performed and consideration has been given other causes, such as allergy and dietary indiscretions.

Lee states that patients suffering from repeated or continuous colds without a demonstrable cause in the nose, throat or chest and in whom there is no indication of allergy or any other known basis for the condition, should have a basal metabolic test. It is certainly true that there are sufficient cases on record of this nature in which the patient's basal metabolic rate is low and where continuous colds have ceased under thyroid medication, to warrant thinking of hypothyroidism as a cause. This is particularly true if the nose presents what might be called a chronic hypertrophic vasomotor rhinitis, which also opens the question of whether iodine medication internally in such cases as, for example, in hydriodic acid, is helpful because of the action of the iodine on the thyroid gland.

Forman's viewpoint is that even if the vasomotor rhinitis is on an atopic basis, it becomes manifest often due to secondary or aggravating factors, and that thyroid deficiency is frequently one of these.

Among the throat and larynx cases, the two most frequent symptoms are that of a dry sensation or of a foreign body in the throat and of a huskiness of the voice associated with vocal fatigue.

That there are ophthalmologists who are thinking about this subject in their practices is attested to by the fact that King and Weiss reported a series of patients whose only complaint was of swelling of the eyelids, but who, upon questioning, presented other symptoms and signs of hypothyroidism, the diagnosis being proven by the reduced basal metabolic rate and by improvement following the taking of thyroid extract. Muscular imbalance of the eyes is not

uncommon in subthyroid individuals, the convergence defects being especially frequently noted.

It is impossible to emphasize too strongly the need for extreme care in the administration of thyroid extract, and it is my firm conviction that progress in the study and care of hypothyroid individuals has not kept pace with that of hyperthyroid patients, largely because required medication has been misused so often. It should be understood first of all that there is no such thing as a standard dosage of thyroid extract. It must be individualized and in two seemingly parallel cases one person may require one-half grain of thyroid per day, whereas another may need six or eight grains or even more daily. Underdosage, of course, brings no results whatsoever, and overdosage plays havoc. Until the proper dosage for the individual has been ascertained careful regular check-ups at intervals of from five to seven days are absolutely essential, care being given on these occasions to a consideration of late afternoon temperatures, pulse reading and the patient's general sense of well-being, which is a most important criterion, with particular questions as to symptoms or signs of overdosage, such as rhinorrhea, unusual palpitation of the heart, nervous instability and sleeplessness.

Contraindications to thyroid medication commonly recognized are hyperthyroidism, diabetes mellitus, serious organic heart disease, persistent tachycardia, tuberculosis and marked undernutrition, although it is well to say in passing that the last named contradindication is an entity only if in a rather extreme degree because, despite all that appears in the literature about hypothyroid individuals being overweight, it has been my observation that many persons badly in need of thyroid medication, as evidenced by their symptoms, basal metabolic rate and subsequent improvement under the medication, have been distinctly underweight and have reached their normal weight only after they have begun to take the thyroid medication. The dual physiology of the thyroid gland adequately explains this.

Some writers have stated that it makes little difference in what form the thyroid extract is given so long as the same form of medication is continued throughout the treatment of a particular case. My preference is definitely for a particular cold-dried extract because I believe that it is better standardized than most of the other preparations offered on the market today. It is, in my estimation, most important to avoid varying the preparation used throughout the treatment of a particular case, and I consider it to be of further advantage to adhere to one particular preparation, if possible, in the

treatment of all of my subthyroidic patients, because I feel that it makes me better able to recognize the point of optimum dosage and particularly the early signs of overdosage.

19 GARFIELD PLACE.

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LXXXVII

DISEASE—OR DEFAMATION?

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Was Beethoven syphilitic? Or is he, as Carl Engel suggests, the victim of those who have tried to fasten on him a disease he never had? An important question—so its increasing discussion seems to indicate. This paper naturally does not attempt to answer it as a physician might, but, with a prefatory statement of the sicknesses which he really had, it does attempt to show the flimsy foundation of the charge; the extent, despite that flimsiness, in which biographers have repeated it, and in brief compass the published opinion of the medical profession. On this basis an answer can be and is arrived at.

For only a short period did Beethoven enjoy the health which with his great natural strength he should have had. For the remainder—from the chills of his childhood trip to Holland, to that moment when the stricken giant shook his fist at the storm and fell back dead—there was a succession of ailments. Smallpox was one of them, for all descriptions include the pockmarks on his face; probably typhus was another. Digestive troubles and chest affections began early in his life at Vienna. In 1798 came the first sign of the deafness which almost drove him to suicide.

By 1810 his hearing, which had been unusually keen, was largely gone and within another half dozen years a silence that was almost complete had closed in upon him. Aggravated by irregular habits, other troubles had in the meantime developed. In roughly chronologic sequence, there came increased digestive troubles with colic; enteritis, bronchitis, followed by chronic catarrh, rheumatism, asthma, pleurisy, eye troubles, intense and long-continued headaches, gout, cirrhosis of the liver (which killed him) with dropsy, and perhaps pneumonia from exposure on that fatal ride from Gneixendorf. The story of the four months' attack that marked the end is too well known to need restatement. In 1817, he had written: "I consider myself doomed. . . . Thank God, I shall soon have finished my part in the play. . . . May God help me." The finish was an appalling struggle for life.

Biographers, not content with this, now quite generally add syphilis. Whether Beethoven had that disease or not is in itself of

little consequence—he had begged that the truth about him, whatever it might be, be told—but it is of consequence whether or not those biographers have without sufficient warrant attributed it to him. Alexander Wheelock Thayer, the great authority on the facts of Beethoven's life, started the theory, strangely enough, through the very vagueness of his statements. What and all that he said in print appears in the following citations—and he said nothing about venereal disease. The inference was easily drawn, however, that Thayer was concealing something and that that something was detrimental or even disgraceful. Syphilis may cause deafness, and Beethoven was deaf; it may contribute to cirrhosis of the liver, and from that disease he died; and Thayer kept something up his sleeve. Therefore, Beethoven had syphilis. To this choice bit of reasoning was added what Thayer was said to have said in letters or orally, what a surgeon of Vienna was said to have said was "probable," and the rumors of two prescriptions.

The charge first got into print in 1879, in Sir George Grove's article on the composer in his "Dictionary of Music and Musicians." Having written, "One thing is certain . . . that he (Beethoven) had no taste for immorality," he says, five pages later: "The whole of these appearances (in the postmortem examination) are most probably the result of syphilitic affections at an early period of his life." The shock comes not from the text but from the footnote:

> "This diagnosis, which I owe to the kindness of my friend, Dr. Lauder Brunton, is confirmed by the existence of two prescriptions of which, since the passage in the text was written, I have been told by Mr. Thayer, who heard of them from Dr. Bertolini."

Thus were these famous but elusive prescriptions started on their biographical career. Some thirty years after Grove, Theodore von Frimmel wrote that, in addition to certain possible causes of Beethoven's deafness, there was another "about which I must not keep silence, since many years ago Thayer gave me by letter definite information about this other malady of Beethoven's." The malady was not named nor the text of the letter given. Then Dr. Leo Jacobsohn said: "After Frimmel's communication I do not feel I am committing any indiscretion when I say that in the private possession of a man of culture there is an as yet unpublished note in Beethoven's own hand referring to a cure that leaves no doubt as to the specific nature of his malady." Hevesy's "Beethoven, the Man" puts it: "In 1819 he (Beethoven) notes: 'L. V. Legunan. L'art de connaitre et de guérir toutes les contagions veneriennes' "—a memorandum that he must get the book, says Ernest Newman in quoting Hevesy. Specht's still more recent "Beethoven as He Lived" (1933) makes

it an order for the book, and gives the price. Friederich Schultze (1928) holds to the original story of the two prescriptions privately held and not to be published.

And now for the beginning of it all, Thayer's statements, here quoted as given in the Krehbiel edition of his "Life of Beethoven." At Volume I, page 253:

"After spending his whole life in a state of society in which the vow of celibacy was by no means a vow of chastity; in which the parentage of a cardinal's or archbishop's children was neither a secret nor a disgrace; in which the illegitimate offspring of princes and magnates were proud of their descent and formed upon it well-grounded hopes of advancement and success in life; in which the moderate gratification of the sexual was no more discountenanced than the satisfying of any other natural appetite—it is nonsense to suppose that under such circumstances Beethoven could have puritanic scruples on that point. Those who have had occasion to ascertain the facts know that he had not, and are also aware that he did not always escape the common penalties of transgressing the laws of strict purity."

And in Volume II, page 294, after quoting from the eulogy written by Dr. Aloys Weissenbach in 1814:

"Remarks follow upon Beethoven's ignorance of the value of money, of the absolute purity of his morals (which unfortunately is not true) and of the irregularity of his life."

And in Volume II, page 87, referring to Dr. Bertolini, Beethoven's physician from 1806 to 1815:

"In 1831 he gave a singular proof of his delicate regard for Beethoven's reputation; supposing himself to be at the point of death from cholera, and being too feeble to examine his large collection of the composer's letters and notes to him, he ordered them all to be burned, because a few were not of a nature to be risked in careless hands."

So stands the whole of the main indictment. Thayer asserts that Beethoven did not always escape the common penalties of transgression, that the reference to the absolute purity of his morals is not well founded, and that a physician had destroyed his correspondence lest some of it fall into careless hands. That is a slim foundation, but it covers all that Thayer committed to print. Not even in discussing the composer's deafness or cirrhosis does he make an intimation of a syphilitic origin. All the rest of the so-called evidence cited by later biographers is at second or third hand, and refers to documents which are not produced and which cannot be published, though the implied existence of the disease is trumpeted to the world! Hevesy and Specht give at least partial specification, but Ernest Closson cleans the slate by saying that the documentary evidence must have been destroyed.

Testimony so indefinite would merit scant attention were it not that other writers have accepted it. That documents which are not produced have no value as evidence is a point which needs little

urging. Even if Hevesy's reference to a memorandum and Specht's to an order were to be accepted, they would only put Beethoven in the position of seeking a book on the symptoms and cure of venereal disease twenty-one years after his deafness had begun and, significantly, at the very time that he was worried to death by a nephew who was given to dissipation but whom Beethoven loved and tried in his groping, unhappy way to protect and educate. This is no attempt to shove disease off onto the nephew, but only to show conditions that should be taken into account. Newman had them in mind, though without acknowledging their import, when he wrote of Beethoven's having "an agonized vision of the pleasure-loving and easily led Carl coming to moral and physical shipwreck in the stews of Vienna."

Mr. Newman, in his book, "The Unconscious Beethoven" (1927), is the most positive of the later writers. "It is a matter of common knowledge that Thayer had in his possession evidence which he could not bring himself to make public." "For about half a century it has been known—or confidently assumed—that some time in his early manhood Beethoven had contracted syphilis, and the further confident assumption was made that this was the ultimate cause of his deafness." He cites Grove, Thayer, von Frimmel, Jacobsohn and Hevesy; brushes aside Dr. Waldemar Schweisheimer who, in 1922, made, as he admits, a "searching examination" of the subject; adds a citation that Beethoven was given to flirtations "from which he did not always emerge happily"—and starts his next chapter, quite unwarrantedly it would seem, with these words of finality: "The fact of Beethoven's malady seems then to be beyond dispute."

The distinguished musical critic realized, of course, the weakness of relying upon unseen documents, but he contends that the accepted rule of evidence does not apply. He says: "That line of reasoning would be valid enough in most cases, but it has to be remarked that the evidence has been withheld not by reason of any desire to secure, as it were, a conviction by suppressing vital facts, but because of an exceptional and commendable feeling of piety towards the great composer." So, without the evidence, and unaffected by the feeling of piety which he thinks commendable in those who could but will not produce it, he finds, and publishes as an established fact, that Beethoven had syphilis. But he is driven to a rather desperate defense—that this matter of evidence is "a question simply of common sense," not a medical question. "We have only to read Thayer's sentences with a perfectly open mind to realize that Bertolini had given him confidential information about Beethoven's disease and

indeed shown him his own prescriptions in the case—the prescriptions referred to by Grove." Here the open mind draws conclusions with a vengeance—that the prescriptions were for syphilis, that Bertolini was the physician who gave them, that the latter had kept copies of them and that they were not among the papers destroyed when he thought he was about to die.

Moreover, Newman makes the burning of the physician's "letters and clinical notes," in 1831, lest they should fall into "careless hands" the basis of an inconsistent argument: "If the documents did not reveal a malady that had not merely physical but moral connotations—which cannot be said of typhus or deafness—could their falling into the most careless hands imaginable do the slightest harm to Beethoven's reputation?" Did then the physician, with his "delicate regard for Beethoven's reputation," burn letters and clinical notes and yet preserve the damning evidence of the prescriptions themselves and more than twenty years later exhibit them to Thayer? "Common sense" seems to cut in both directions in this matter. If Dr. Bertolini burned anything with the object of preventing the world from knowing that Beethoven had syphilis, he certainly burned the prescriptions, if there were any, and could not have shown them to Thayer or anybody else.

Dr. Bertolini, after mentioning Julietta Guicciardi, Bettina Brentano and Mme. von Frank, is reported to have added: "In addition he sought favors elsewhere, too, but that did not always agree with him" ("from which he did not always emerge happily," is Newman's translation). There is no intimation, so far as has been revealed, in anything that Dr. Bertolini left, of the nature of the "favors," or even that they differed from those which the composer received from the high-born ladies named, unless from the phrase that they "did not always agree with him," which might mean any one of a number of things.

Having, without the production of the evidence, established Beethoven's guilt to his own satisfaction, Mr. Newman seeks to sustain the verdict by inferences drawn from incidents of his life—these inferences, however, being manifestly the outgrowth of the syphilitic theory and not its proof. Beethoven's malady, he says, will "account for his venomous hatred of unchaste women and for his desire to protect his brothers from them"; will explain the "excessive restrictions upon the goings and comings, the companions and the amusements" of the nephew, the "unwarrantable interference" with the lives of his brothers and the "hatred of their women-folk that passed the bounds of reason."

The "desire to protect his brothers" was expressed in 1796, in just eight words. The composer had gone to Prague, and from there he wrote to Johann: "I hope you will enjoy living in Vienna more and more—only beware of the whole tribe of bad women." If, as actually suggested by Newman (p. 45), and by several others, this wise advice to a youngster just turned loose in the gayest capital in Europe is a symptom of venereal disease, then a father who seeks to guide his son is under suspicion, and the author of the seventh chapter of Proverbs is beyond salvation. As to the "unwarrantable interference" with the brothers' lives and the "hatred of their womenfolk" the record shows Beethoven's judgment of the two ladies who became his sisters-in-law to have been correct, and his attempt to protect his brothers' welfare, the family name and, indirectly, his own high connections in Vienna, to have been justified in purpose, if not in the wisdom of the means adopted. Johann's wife, Therese Obermayer, had been his housekeeper and mistress. She brought her husband an illegitimate daughter as a wedding gift, says Herriot, and Specht adds that her conduct exceeded Beethoven's worst fears. Thayer records that her conduct reached the "extreme of reprehensiveness" during a spell of sickness which threw her husband on his back and "the woman chose this time to receive her lover in her house and to make a shameless public parade of her moral laxness." Newman himself says: "Therese turned out to be a rather worthless character whose infidelities were notorious." Of Carl's wife Thayer said there could be no doubt that she "was wicked and vicious." After her son's birth, she had an affair with a medical student, robbed her husband and was sent to jail for a month. While her husband's corpse was still in the house, she passed the night with her lover. Mr. Newman concedes (he could scarcely do else) that the lady's "morals were not what they might have been," but asserts that Beethoven's taking control of the son was a "breach of a solemn trust," referring, of course, to the codicil of the brother's will. A difficulty here is that the highest court agreed with Beethoven and that its judgment was sustained on appeal to the Emperor. To this Mr. Newman's only answer is that the court "perhaps was not uninfluenced by the fact that Beethoven was known to be the protege of the Archduke Rudolph."

That the treatment of the nephew, over whom Beethoven agonized for years, should be ascribed to syphilis seems beyond justification. "There is nothing in the narrative which brings reproach upon Beethoven so far as his high sense of duty and disinterested affection for his nephew are concerned," wrote Thayer, and, though deploring the consequences of that unwise attempt at guardianship upon

Beethoven himself, the author added: "He was proud of the boy's intellectual gifts, which were out of the ordinary; he dreamed of seeing him great and respected in the eyes of the world; he wanted loving companionship now and in his old age; he hungered for sympathy and for help . . .; he desired to see by his side and in his kin an incarnation of that polite learning and that practical knowledge of worldly affairs which had been denied to him. All his aims were laudable, all his desires natural and praiseworthy; but he was the last man in the world to know how to attain them." As to the restrictions "upon the goings and comings, the companions and the amusements" being "excessive," Johann asserted that Carl knew every strumpet in Vienna, and, said Holz, Beethoven's constant companion for several years—vital years so far as the nephew was concerned—"Investigation proved that Johann was right." Severe restrictions seem to have been in order.

Mr. Newman makes a point of Beethoven's own marriage as a factor leading up to a period of "bitter hypochondria," and says: "His disease, barring him, the most affectionately domestic of men, from marriage, must have been, apart from its physical distress, a sore trial to his spirit." If the composer was barred by syphilis from marriage, he certainly never knew it. He proposed to Magdalena Willmann, courted Julietta Guicciardi, was engaged to Theresa von Brunswick, asked for the hand of Theresa Malfatti, was seriously in love with Amalie Sebald, and wrote the famous and poignant letters to "The Immortal Beloved," whose identity has remained a mystery, but with whom, as the letters bear testimony, he was in the relationship of declared and accepted love.

The effect of his own vehemence upon Mr. Newman is curious. In the *London Sunday Times* of November 27, 1932, set off by Edgar Lee Master's poem "Beethoven's Ninth Symphony and the King Cobra," he emits this harsh comment: "The Beethoven case is one not merely of the gradual conventional falsification of history and biography as the years have rolled on but also of a conventional falsification of criticism." He invokes a devil's advocate, timed a hundred years hence, and makes him address Beethoven in words intended presumably to register the final verdict of mankind: "In a rational artistic world you would be prosecuted for having obtained sympathy under false pretenses. Whether you intended it or not, you have put up the biggest bluff in musical biography, and thanks to the incurable sentimentality of the eighteenth and nineteenth centuries, you succeeded in getting away with it." And so on for a column. "So the cold indictment might run," concludes Newman, "and prob-

ably shall run in the next generation but one." An ardent follower, Alan Pryce-Jones ("Beethoven," 1933), made it the "duty" of his book "to correct" the legends of Beethoven's life. With an expression of affection "in the shelter of a century's distance," he pictures the composer—"loutish, dirty, ungrateful, full of deceit, of lies, of treachery, of whining complaints; ugly, snobbish, hypocritical"—with the astonishing conclusion that "the common, barren, drab life of Beethoven was building something of which he himself was never aware." As authority he cites Mr. Newman's article.

The music critic has at any rate achieved the honor of being cited as a medical authority in a medical magazine. Dr. Ira Frank, of Chicago, in THE ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, for June, 1935, says that Newman has made it "clear" that the composer suffered from syphilis.

Upon the appearance of Newman's book, an English musician with some medical training, William Wallace, jumped to his support in *The Musical Times*, of June 1, 1927, with evidence which, he asserted, "clinched the diagnosis." As early as 1914, Wallace had pronounced upon Beethoven's illnesses. "The cause was syphilis," he said in his book, "The Musical Faculty," and proceeded: "From first to last, Beethoven's clinical history illustrates the usual course that his malady takes." In 1927 came his "clinching" proof, a "certified photograph" of Beethoven's skull taken at the exhumation of the body in 1863, showing the bone in the region of the right ear to be "enormously thickened." A photograph is a skittish thing, at best, but there is here also the question of what it was that was photographed. At the autopsy the organs of hearing were dissected, and Dr. Frank says they "were preserved in the Anatomical Museum in Vienna, but when, many years later, Politzer desired to examine them for study, they had disappeared." This is confirmed by a letter from Dr. Politzer to Dr. Cabanes, February 12, 1901. It is further recorded that at the exhumation, in 1863, the skull was found to be in nine pieces, and Gerhard von Breuning, who as a lad had waited at Beethoven's death bed and was now a leading surgeon in Vienna, had the sad duty of reconstructing it. "The skull was completely distorted by the dampness of the grave," says Paul Bekker, and Ernest Closson adds that certain parts proved to be missing. Of the photograph taken by Rottmayer and preserved in at the Beethoven Museum in Bonn, Dr. Sorsby says:

"No such bulge is shown in the masks of Beethoven taken during life and on his death. The postmortem report speaks of the uniform thickness of the vault of the skull. The careful description of the fragments disinterred in 1888, when they were in very much the same condition as in 1863, makes no mention of any irregu-

larity in the bones, and Choulant's drawings do not show it. The bulge in the Rottmayer photography—and enlargements based on it—is therefore regarded as an artifact; probably a piece of clay attached to the bones."

Dr. Choulant's drawings were made upon the second exhumation, at which time there was a second reconstruction of the skull, and Dr. Georges Canuyt, in a discussion of Beethoven's deafness (*Le Medecine International Illustree*, March, 1934), asserts that Drs. Weisbach, Fold and Meinert took exact measurements of the skull and did not report any thickening of the bone. Mr. Wallace's confident pronouncements seem to have been without foundation.

The influence of Mr. Newman's skillful writing upon other commentators has been marked. Daniel Gregory Mason, distinguished musicologist and composer, while refusing to accept the picture "more like a caricature than a portrait," presented by the "more aggressively 'modern' writers" ("Beethoven and His Forerunners," 1930), takes Newman at face value, and refers to Beethoven's "well known hatred of loose women, . . . a phobia due to his unconscious association of sexual irregularities with the venereal disease which in his case they had led to, and with the terrible affliction of deafness which that had brought in its train" ("Essays," 1928). Former Premier Edouard Herriot (La vie de Beethoven," 1928) puts a query: "One must ask oneself if he did not also suffer from another malady very widely distributed in the Vienna of that epoch, as we are told, and less easily treated then than today." Romain Rolland ("Beethoven the Creator," 1928) says "perhaps from syphilis" and refers to "an excess of scrupulosity" which "prevents the publication of certain documents that exist in a Berlin collection." W. J. Turner ("Beethoven: The Search for Reality," 1927) is in opposition to the extent of citing the composer's ability to hear through bone conduction as against a syphilitic origin of his deafness. Marion M. Scott ("Beethoven," 1934) asks, apropos of the injunction to Johann to beware of the whole tribe of bad women, "Had Beethoven become the burnt child who dreads the fire?" and again, as to the Heiligenstadt Testament: "The disaster was as complete as any in a Greek tragedy, and if biographers are right who believe that Beethoven's deafness resulted from a syphilitic trouble, then it also fulfilled the Greek doctrine of Nemesis." J. H. Elliot ("Music and Letters," January, 1934) refers to the "unsavory explanation of Beethoven's ultimate deafness." René Fauchois, in his often unpleasant and imaginative "La Vie d'Amour de Beethoven" (Paris, 1928), asks: "Was Beethoven affected by syphilis? Was it for that that he was denied experience of all that comprises the ordinary happiness of mankind? . . . Certain documents exist, it appears, in a collection in Berlin, details which Beetho-

ven transmitted to a physician who was treating him." Pryce-Jones says: "A high proportion of the great has been syphilitic, and although his deafness gave no evidence of syphilis, Beethoven may well have been among them." Then he gets a bit stronger: "His behavior to his sisters-in-law is probably rightly diagnosed by Mr. Newman as the result of a sexual complex due to his own disease." Robert Haven Schauffler ("Beethoven, the Man Who Freed Music," 1929) concludes that Beethoven probably suffered from a venereal disease, that the cirrhosis may have been so caused, that the deafness was not, and that the "hatred of the sisters-in-law had at times the blind intensity of a sexual obsession." Chotzinoff ('E'roica," 1930) has syphilis running as an undercurrent—not named—in his romance on the composer's life. Hevesy ("Beethoven the Man," 1927) darkly questions, "or from some still more formidable malady?" and puts in a footnate: "The poison which was undermining his body was day by day destroying his hearing." Ernest Closson ("L'élément Flammand dans Beethoven," 1928) disposes of the missing papers: "It is generally admitted today that the musician suffered from a secret malady, not hereditary but directly contracted, upon which the piety of his friends and his biographers has drawn a veil and the documentary evidence of which must have been destroyed." Finally, a writer in *The Musical Times* refers almost casually to the "usually accepted" cause of the deafness.

These subsequent writers in the main follow or agree with Newman; Newman relies upon Thayer—"A biographer of Thayer's exceptionally judicial habit of mind would not have said all he did say on such a matter unless he had been absolutely sure of his facts." He quotes Grove's statement above given with the footnote regarding Dr. Brunton, discusses Dr. Schweisheimer who opposed the syphilis theory, refers in a footnote to Dr. Jacobsohn on the cause of the deafness—and so far as concerns medical authority he stops right there. Succeeding writers in general make no references whatever to medical diagnosis. In other words, the new school of biography rests its case upon Thayer's cryptic utterances and the alleged incriminating documents which have not been produced in the fifty-seven years that have elapsed since Grove's article was published. Without medical support for its assertions, the conclusion would seem inevitable that, whatever virus coursed through Beethoven's body, the virus of an inadequately supported charge is poisoning his biography.

The medical profession in the meantime has shown a constantly increasing interest in Beethoven's case, with especial reference to his deafness. Dr. Beverley R. Tucker ("The Gift of Genius," 1930)

diagnosed it as due to infection of the middle ear, but the infection was not syphilitic. The author says:

"It has been hinted that Beethoven's deafness and idiosyncracies were probably due to syphilis, but no evidence, clinically or from the autopsy of his brain, can be adduced of the disease. From all descriptions he had middle ear deafness, cirrhosis of the liver and ascites (dropsy), and in all probability the whole vicious circle was inaugurated by nasopharyngeal infection early in life. Again, Beethoven's mental or rather temperamental aberrations were not those of paresis (syphilis) but in reality those of congenital emotional instability aggravated by circumstance and by deafness."

In the book written by Dr. G. Bilancioni ("La Sordita di Beethoven," 1921) syphilis as a cause is not suggested, and Dr. Schweisheimer is quoted to the effect that is it not mentioned in connection with Beethoven by any authority. Dr. Bilancioni's conclusion was this:

"Beethoven's deafness was caused by otosclerosis, that is, by a disease intimately connected with disturbances in the calcium metabolism which occur in serious organic changes found in people with alcoholic and tuberculous antecedents."

Dr. Schweisheimer (1922) denied that the autopsy showed any condition probably referable to syphilis, and rejected the contentions based upon the alleged thickening of the skull. Dr. Marage, of Paris, in an address to the Academie des Sciences, in 1928, and in subsequent correspondence with Rolland, also eliminated syphilis, a disease which "produces an absolutely different order and development of deafness." Raoul Blondel ("The Deafness of Beethoven, London Quarterly Review, January, 1931) commends Dr. Marage as especially qualified and cites his researches as leading to a diagnosis of inflammation of the inner ear caused by a grave pathologic heredity, abuse of alcohol and liver trouble. Dr. Maurise Sorsby, of London, (*Journal of Laryngology and Otology*, August, 1930), finds no indication that syphilis was the cause either of the deafness or the cirrhosis. The alleged circumstantial evidence that Beethoven had the disease, he adds, "is hardly deserving of serious attention." That otosclerosis is commonly caused by syphilis, as Jacobsohn contended, Dr. Sorsby declares is "an exploded fallacy," and he adds: "Cirrhosis of the liver of syphilitic origin is as questionable an entity as otosclerosis of syphilitic origin. There are quite enough causes in Beethoven's life for cirrhosis without having to call in a problematic syphilitic infection." Dr. Henry L. F. A. Boyle, of Paris, also rejected the syphilis theory ("Beethoven vu par les Medicins," 1928).

Dr. Thomas J. Harris, in a paper read at the New York Academy of Medicine (1936) reached the conclusion that "syphilis involving the ear is not characterized by such symptoms as Beethoven had,"

and that the cause was otosclerosis. Dr. Jacobsohn (1910) had also said otosclerosis—probably the result of typhoid fever but possibly of syphilis—this latter on the basis of the alleged thickening of the right parietal bone: "A very feeble argument," said Canuyt, "since every infectious disease may cause the same lesion and produce a like result." Jacobsohn's reference to the alleged note mentioning a remedy "the use and purpose of which are not doubtful," met a similar fate in Canuyt's article: "This is of little consequence. Beethoven had consulted so many physicians and taken so much medicine that even if there should be found a prescription directing the use of mercury, it would be nothing extraordinary." Canuyt attaches no value to unpublished documents, but seems to follow Professor Escat in the opinion that the disease may have existed in hereditary form, and he is "inclined to hold" that the deafness was of labyrinthine type, and of syphilitic origin, whether inherited or acquired.

Dr. Ira Frank, in his paper above cited, approaches his subject in fine spirit: "We must stand reverently before this Prince of musical geniuses. . . . Beethoven was not alone a great musical genius, he was also a great man." Unfavorable conditions are listed, and then comes the "possible cause" syphilis, though other causes "seem more probable." At this point Dr. Frank seems too credulous, for he says that Ernest Newman's "The Unconscious Beethoven" makes it "clear that the composer suffered from syphilis, for the treatment of which he received prescriptions (still in existence) which relied on the specific action of mercury." This is a startling statement. Newman makes no reference to mercury, does not claim to know the contents of the alleged prescriptions, and does not say that they existed in 1927 when he wrote his book or at any other time. All Newman asserts is that somebody else said they had existed. The point being novel and of importance, inquiry was made of Dr. Frank and he was good enough to reply that he had secured his information about the mercury from Mr. Felix Borowski, of Chicago, the musical critic. Mr. Borowski, in answer to further inquiry, could only rely upon his memory, as his books and papers were in storage, but he kindly stated his recollection under date of February 1, 1936: "I did indeed give to Dr. Frank the information concerning the prescriptions containing mercury or some mercurial preparation. I am under the impression that I saw a copy of the prescription in a German paper a number of years ago, and it is possible that I made a note of it for future reference." The matter of the prescriptions stands, therefore, as it did before, with contents unknown and in the custody of a person undisclosed. Mercury is an easy enough guess, but it was used in Beethoven's time for a long list of diseases.

Of medical authorities, Dr. Frank cites Klotz-Forest (whose diagnosis is catarrh of the eustachian tube developing into otitis media); Baratoux and Natier (progressive labyrinthine spongification with ankylosis of the stapes); Garnault (atrophy of the nerves due to inflammation); Bonnier (a common tympanolabyrinthine sclerosis); Marage (labyrinthitis from cerebral and intestinal causes); and Bilancioni (otosclerosis). He adds: "All the evidence appears to favor Bilancioni's opinion and with it one is forced, in the absence of more decisive proof of any other condition, to be in agreement."

"It is indeed on slight grounds that the attribution of syphilis has been made," said Dr. J. F. Rogers (*Popular Science Monthly*, March, 1914). Perhaps Mr. Newman has after all hit upon the proper characterization. "*Beethoven's suppositious syphilis*," he calls it on page 39 of his book—and the supposititious is by definition the counterfeit, the spurious, or, even at its best, the hypothetical—a thing assumed without proof.

Mr. Newman must shoulder responsibility for much of the present day assumption that Beethoven had the disease, and—against the preponderance of published medical opinion—that he was deafened by it. In recent months the composer has been made a classic instance. Francis Watson's "Life and Times of Catherine de Medici," telling of Lorenzo's marriage to Madeleine de la Tour d'Auvergne, quotes a contemporary comment: "When she married the Duke of Urbino, she did not marry him alone, for the pox also was her bridegroom." The author points the moral thus: "As we read these words, the grinning skull detaches itself from the gaudy Renaissance trappings. . . . The lilies of Florence gleam on his embroidered surcoat, but poison riots in his veins, the poison that darted its serpentine colors of death through the shining marble edifice of the new age. . . . It was destined . . . to deafen Beethoven." Thus is biography developed.

"When at length Beethoven walked in eternal silence," wrote Dr. Sorsby, "there came to him the mightiest of all cataracts of sound; it could hardly have mattered to him that he missed the noise of the street and the clamor of the market, for the gods had given him to hear and to hand down the Ninth Symphony."

The consideration of those closing years of unexampled creative power tempts to a positiveness rivaling that of the biographers. To the query posed in the title of this paper, the answer, now at any rate, is—It is defamation.

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LXXXVIII

PROPHYLAXIS IN PARASIGMATISM*

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There are, at least, more than four million lispers in the United States alone, the lisping of the majority of whom might have been prevented. The laryngologists, speech clinics and schools are already doing considerable for the lisper, which is amply demonstrated by the fact that there are almost six times as many lispers in the first grades of school as in those beyond the third year.

Scientists have called the production of a distinctly different sound from that desired or the constant substitution of one letter for another, *paralalia.* I will discuss one type of paralalia, *parasigmatism:* the inability to pronounce the letters *s* and *z*, or the substitution of other consonants, such as *t*, *d*, or *th* for them, or their complete omission in utterance. And for further definition, I will devote this paper to the prevention in particular of these *s* and *z* sounds.

The first subject to be considered is how the *s* and *z* sounds are made, and how they differ. By the *s* element, I refer to the sounds in "sit," "cycle," "psychiatry," "scene," "miss," "mouse," "mice," etc., and not in "shoe," "issue," "vision," "was," etc. By the *z*, I refer to the sibilants in "zoo," "does," "buzz," etc., and not those in "azure," "leisure," etc. In articulating the letter *s* there are three important considerations. In order to produce a sound that has the extremely high pitch of the *s*, which ranges from three to four times as high as the extreme right-hand note on the piano, it is essential to have the air blow past the sharp edges of the hard teeth. Second, the column of air must be very fine and swift. Third, there must be no soft surfaces near so that the vibrating speed of the air will be slowed down. For the first consideration, the teeth must be closed edge to edge. In order to do this, it is necessary for the lower jaw to be moved slightly forward so that the teeth will close as in biting. For the second factor, it is necessary to have a narrow groove down the middle of the tongue, and to have the tongue otherwise against the roof of the mouth. There is a slight groove also in the gum ridge above and behind the upper teeth which with the tongue groove will make a

*From the Speech Clinic of Dartmouth College.

cylindrical aperture for the fine column of air which is to be directed through the small opening which remains between the teeth after they are closed together. For the third point to keep in mind, the lips must be drawn back widely and be open, as in smiling, or showing one's teeth. This is the position the lips are in when one says the vowel sound in the word "eat." The *z* sound is articulated identically the same as the *s* except for one thing. In the case of the *z*, the vocal cords in the larynx, or Adam's apple, are vibrating, but when saying *s* they are not. Thus the *z* is voiced and the *s* is unvoiced.

It is important to keep in mind the correct way to say the *s* and *z* sounds when attempting to teach them to a pupil or correcting them when defective. However, it is most important to remember that the speech mechanisms of different individuals differ as much as do their faces. A trained person could recognize various individuals by simply looking into their mouths. Although the principles underlying the correct production of the *s* and *z* sounds must be carried out, individual differences must be considered. After all, it is only the resulting sound that we hear, and not how it was made that designates it as a *s* or *z*. So it really does not matter how one articulates, so long as they produce the correct result. Of course it is well to have the production appear as natural as possible and to have the movements not seem peculiar.

Many children, when they first start to school, have never said a *s* or *z* sound. Many have never mispronounced these sounds; they have simply omitted them. In order to protect them from a permanent embarrassing speech disorder, they must be trained to produce the sounds while yet within the pliant age. The teacher should demonstrate a correct model for the child's imitation, by exaggerating the lip position and biting the teeth together. It must be remembered that there is a distinct difference between the *s* sound and the name of the letter S, "ess." After repeating the *s* sound several times the teacher will find that the child has already assumed the correct position imitatively, and will be able to say the sound after her when so directed. It is often well to call the sound a hiss, or give it a simile, like the sound of escaping steam. After the child has mastered the *s* sound, the teacher can train its incorporation into speech by prolonging it initially in words, as s-s-so, s-see, etc. It can then be lengthened for final and medial acquisition, as yes-s-s, dus-sty, etc. The *z* sound can then be taught likewise, and it is well to compare it to the buzz of a bee, z-z-z-z. When the *s* is defective, the *z* is usually poor likewise. The child can learn the difference between the *s* and *z* by

placing the fingers over the teacher's throat when she says the two, and then over his own in imitative pronunciation.

A child sometimes produces the mistaken pronunciation of the *th*, as in "thick," when attempting to say the *s*, as in "sick." The prevention of the lisp in this case is merely a matter of training the child to bite the teeth together, and in this way a removal of the tongue from between the teeth becomes a necessity. All speech training should be concerned with only isolated speech elements, which, after mastery, can be included in the utterance of words. It is best for the teacher, who expects to re-educate a child's speech, to examine her own enunciation first by means of a hand mirror. The corresponding *z* defect in the above *s* disorder is the pronouncing of the *th* as in "though" for the *z* in "zone." These defects are called frontal lisps.

A *sh* sound as in "show" is often substituted for the *s* in "so." In this case, if the retracting of the protruded lips to the smiling position does not correct the sound, the child must be taught to move the tongue farther forward so that a smaller stream of air will be directed past the teeth. When the *z* is pronounced as in *zh* in "fusion," similar procedures, although voiced, must be negociated.

A commonly encountered *s* disorder, which is very difficult to correct, is the phonetic substitution of *t*, as in "tea," for the *s* sound in "see." The *s* is then taught as a new sound in isolation, and the differentiation between the continuity of the *s* and the explosiveness of the *t* must be learned before the new sound is incorporated into words. The child must be taught to prolong the *s* element greatly, and gradually shade it into the vowel without impeding the sound continuum. The defective pronunciation of *d*, as in "do," for the sound in "zoo," is the voiced equivalent. Sometimes there is not simply a substitution as described but the *t* either precedes or follows all *s* sounds. However, in this case the diagnostic speech retraining proceeds likewise as above.

When the *s* or *z* sounds are discovered to be nasal, the *z* resembling *n*, as in "new," the child must first be trained to blow through the mouth. It is wise to have the child actually direct the air column to some purpose, such as the blowing out of a candle, or the causing of a strip of paper to waver. Then the *s* and *z* articulation can be built on this oral breath stream. These disorders are referred to as nasal lisping.

When a child's front baby teeth drop out, naturally a lisp of somewhat the *th* quality results. This mispronunciation must be com-

pletely disregarded until after the two front permanent teeth are developed. Many lisps have resulted from impetuous speech training during the change in dentition. However, immediately after the two front permanent teeth are present, and this refers simultaneously to the presence of both the upper and lower set, a correct *s* and *z* standard must be established. When the teeth on either side of the two in front are missing, it is quite easy for the air to pass out on either side of the tongue. This is what is called the lateral lisp. This must not be tolerated, if a permanent disturbance of the most severe type is to be prevented. The child must be trained to direct the air flow down the middle of the tongue, even if a toothpick, placed between and below the upper teeth and back along the midline of the tongue has to be used. Jewish children are very frequently lispers. This is often the result of no management after these permanent teeth are established. The Jewish child needs special attention in this respect, because of the characteristic loss in hearing acuity on the high *s* pitches. Unless prevented, they may develop a frontal, lateral or *sh* lisp.

It is easily observed that the soundness of the teeth is essential to the prevention of lisping. Everything should be done in the matter of diet and hygiene which will insure normal dentition and no dental decay. Milk is of cardinal necessity. Vitamins, proteins and minerals are most important for the teeth, rather than carbohydrates. In the matter of diet and hygiene in this connection the dentist and doctor should be consulted.

However, there are abnormal conditions which must be taken into consideration, such as teeth growing out of place or in crooked position, which should receive dental or orthodontal attention.

When the upper front teeth are separated some distance, the column of air must be much smaller and swifter, and be directed more to one of the teeth up high on the side, near the gums, or at the biting edge.

When the upper and lower teeth do not naturally bite together, which is called a malocclusion of teeth, the child must be trained to move the teeth together as in biting, when attempting to produce the *s* and *z* sounds.

However, quite often in these children the malocclusion is so great, the lower teeth so far in front or in back of the upper, that they cannot be brought together to bite. Also there is the condition, called the open-bite, in which, when the back teeth come together, the angle of the jaw prohibits the front teeth's meeting. In these

conditions the tongue must be pressed very hard against the roof of the mouth, and very far forward, so that a fine column of swift flowing air is directed against the lower part of the upper teeth. The sound, in this case, acts as the control. The teacher can imitate and better teach this articulation by placing her fingers between her own molars and learning to give a correct pronunciation of the *s* and *z*.

Through accident, teeth are occasionally found to be missing. If those teeth are normally concerned in *s* and *z* production they must be substituted in speaking by the surrounding teeth, or dental reconstruction must be resorted to. The rehabilitation of the *s* and *z* disorders resulting from such a condition can be negotiated by twisting the tongue so that the air is directed past teeth remaining at either side. However, only one side should be so utilized.

There are individual differences, which are quite permissible, in the production of the *s* and *z*, which are due to dental peculiarities. As mentioned above, there is usually a slight hole between the front teeth at the bottom resulting from the slightly rounded corners of the teeth. If there is no such space, the child will minutely separate the teeth for the utterance of these sounds. Other chips and grooves in the lower edge of these teeth cause the air to be directed through them. The same phenomena, of course, is true of the lower teeth. Thus it is obvious that people can take advantage of their own natural anatomic mechanism, and although they produce the sounds correctly, they do it individually.

There is an unfortunate situation, abominated over a hundred years ago, by the father of the Bell who invented the telephone: a sort of social maladjustment which today still disturbs speech. Sometimes young girls, and even grown women, quite consciously will assume a lisp, believing that by so doing they gain a cute or romantic effect. This can only be obliterated by their persuasion of their own foolishness in the eyes of those they had wished to impress.

The Scientific Papers of the American Bronchoscopic Society

LXXXIX

ADDRESS OF THE PRESIDENT*

H. Marshall Taylor, M.D.

Jacksonville, Fla.

Fellow Members and Guests of the American Bronchoscopic Society:

To serve the American Bronchoscopic Society as its nineteenth president is a privilege. For the signal honor accorded me I am deeply grateful.

In a presentation before the American Laryngological Association last year I discussed the ossification of the cartilages of the larynx and its relationship to some types of laryngeal disease. Since that time my interest in this subject has been enhanced, and I feel that I may best serve the society at this time by the further consideration of this phase of our specialty.

The popular conception of age as the one etiologic factor of ossification of the cartilages of the larynx is misleading, for numerous other factors appear to have etiologic significance. Senile change was for centuries regarded as the sole causative agent, but anatomic writers of the nineteenth century recognized the wide variability of the age period at which ossification of these cartilages occurs and distinguished between the pathologic process and the process which is to a certain extent normal. The consensus is that, in general, the ossification of the laryngeal cartilages advances with age, but so great is the variability of the age period that it is impossible to determine the age of persons from the degree of ossification. That the process has been found in a youth twelve years of age and has not been discernible in a man aged 103 years indicates the wide variation. Investigations pertaining to the age factor have resulted in the conclusion that actual bone formation takes place before full maturity is reached, thus confirming the opinion of an early anatomic writer

*Read before the nineteenth annual meeting of the American Bronchoscopic Society, Detroit, May 27, 1936.

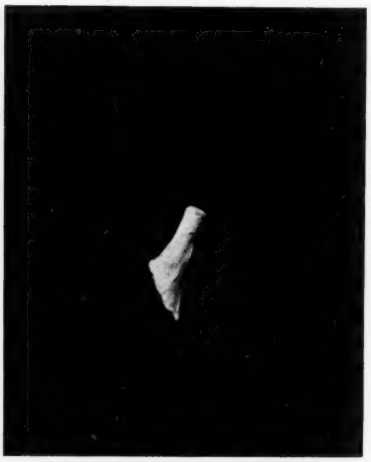


Fig. 1. Case 5. Superior cornua of thyroid cartilage after expulsion.

that ossification begins normally when skeletal growth is otherwise complete.

In men, after the age of fifty years, the process is well advanced in the cricoid cartilage and usually complete at the age of seventy; in the thyroid cartilage after the age of forty the ossification of the alæ is almost complete; but the osseous change in the arytenoid cartilage progresses so gradually that it is complete only at an advanced age. In women, however, the process in all three cartilages advances more slowly and at all ages is less extensive than in men.

The conclusions of the anatomists have to a great extent been confirmed by the findings of the roentgenologists, although the former group agreed that ossification begins first in the cricoid, next in the thyroid and last in the arytenoid cartilage, and the latter group found that in general the process occurs in the thyroid earlier than in the cricoid cartilage. Both agreed that the process advances with age, even though it is impossible to determine the age of persons from the degree of ossification. Also, both noted that it progresses more slowly in women than in men and that in the thyroid cartilage it progresses differently.

The value of roentgen examination of the larynx as a means of studying the process of ossification in the laryngeal cartilages was first recognized in 1896, but it was five years later before this means of studying the process in the living was advocated. Numerous roentgenologists as well as anatomists noted the wide variability in the

Figs. 2 and 3. Case 1. Roentgenogram and drawing showing dense deposition of bone in thyroid cartilage. Syphilis tends to increase the intensity of ossification which usually occurs in irregular bands extending in all directions. This patient gave a syphilitic history and a positive Wassermann.

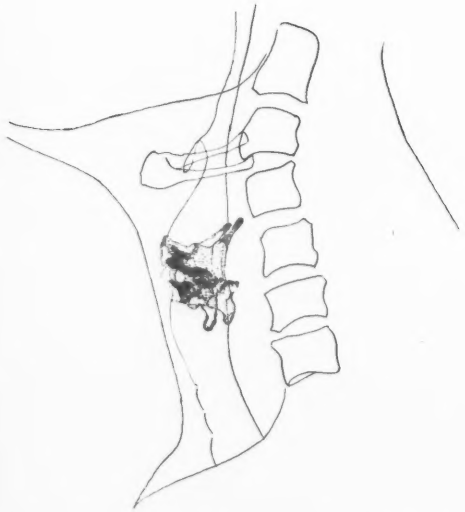
rapidity and degree of change, and because of this variability one writer likened the process to the graying of the hair.

The beginning calcification and progressive ossification which follow the attainment of sexual maturity render the laryngeal cartilages visible in the roentgenogram. In the cricoid cartilage the process advances from the upper lateral border and the region of articulation with the thyroid cartilage, the border adjacent to the thyroid cartilage showing ossification before that adjacent to the trachea. In the thyroid cartilage the development of the process differs in the two sexes. After advancing upward from the region of the inferior cornu along the posterior border in both sexes, it develops in men after about the age of thirty years, from the cornua forward along the borders until the areas of bony tissue meet and thus form round or oval windows of cartilage, which become progressively smaller until the whole cartilage may undergo complete ossification; in women it advances through the entire width or may take place by degrees in various areas. Extensive ossification of this cartilage may occur in young persons and only a limited degree in older ones, but usually at the age of fifty to sixty years both the shape and the structure of the bony tissue are shown in the roentgenogram. In the arytenoid cartilage the centers of ossification appear first at the base and proceed upward to the apex.

Early writers as well as more recent investigators mentioned the lack of evidence of osseous change in the epiglottis, the corniculate and the cuneiform cartilage. There is ample evidence to show that these cartilages of the elastic variety do not tend to ossify as rapidly as those of the hyaline type, such as the thyroid, cricoid and arytenoid cartilages.

As a rule, the initial processes of ossification are found at the points of insertion of the ligaments or muscles in the laryngeal cartilages. Consequently, on theoretical grounds, some writers were of the opinion that the advancing calcification and ossification of these cartilages affect the voice, especially the singing voice. The question of muscle pull on these cartilages and its possible relation to ossification, especially in singers and public speakers, is suggestive enough to invite further investigation.

Pathologic processes of ossification occur in the larynx in three forms: ossification in cartilage tumors of the larynx, excessive bone formation in the form of exostoses or bone plates formed in cartilage rests in the mucosa, and premature and excessive bone formation in the laryngeal cartilages in chronic infectious processes. One early



Figs. 4 and 5. Case 2. Roentgenogram and drawing of the thyroid cartilage showing a marked ossification, only an oval window remaining of cartilage. Chronic infections predispose to excessive bone formation in the laryngeal cartilages. The patient, a woman fifty-five years of age, had suffered from a chronic pansinusitis for many years.

anatomic writer stated that normal calcification of the laryngeal cartilages results from the increasing vascularity of the larynx following puberty, that vascularity of these cartilages is pathologically increased in chronic inflammatory processes, and that calcification may, therefore, occur prematurely and extend more rapidly than normally.

Some observers found that tuberculosis tends to delay ossification. In cases of tuberculosis of the larynx, a tendency to "demineralization" of the laryngeal cartilages was noted by two investigators, who regarded it as related to general decalcification in this disease. They stated that in such cases scattered denser areas of ossification may be observed, especially in the lower part of the thyroid cartilage.

They also found that in syphilis of the larynx the roentgenogram shows an abnormal degree of ossification in the lower posterior border of the thyroid cartilage, occurring in irregular bands and extending in all directions. Others noted that this disease tends to increase the intensity of ossification and to lead to the formation of hyperostoses and osteophytes.

Carcinoma of the larynx appears to hasten the process of ossification where the growth is not so rapid as to cause early destruction of the cartilages. Frequently they are destroyed, but from the perichondrium new cartilage and bone tissue may be formed, which appear in the roentgenogram as scattered areas of calcified and ossified cartilage.

There is danger that a normal area of ossification may in roentgenograms be mistaken for a foreign body. In discussing my paper last year, Dr. Chevalier Jackson stated that he had heard the point made in differential diagnosis of ossifications in the laryngeal cartilages and bones lodged as foreign bodies in the hypopharynx or upper esophagus, that these bones are always seen at a lower level than the isolated spots of ossification which may be mistaken for bones. This general rule he found to be true in 90 per cent of the cases, but he recalled several striking examples of bones occurring as foreign bodies exactly at the level of the cricoid cartilage.

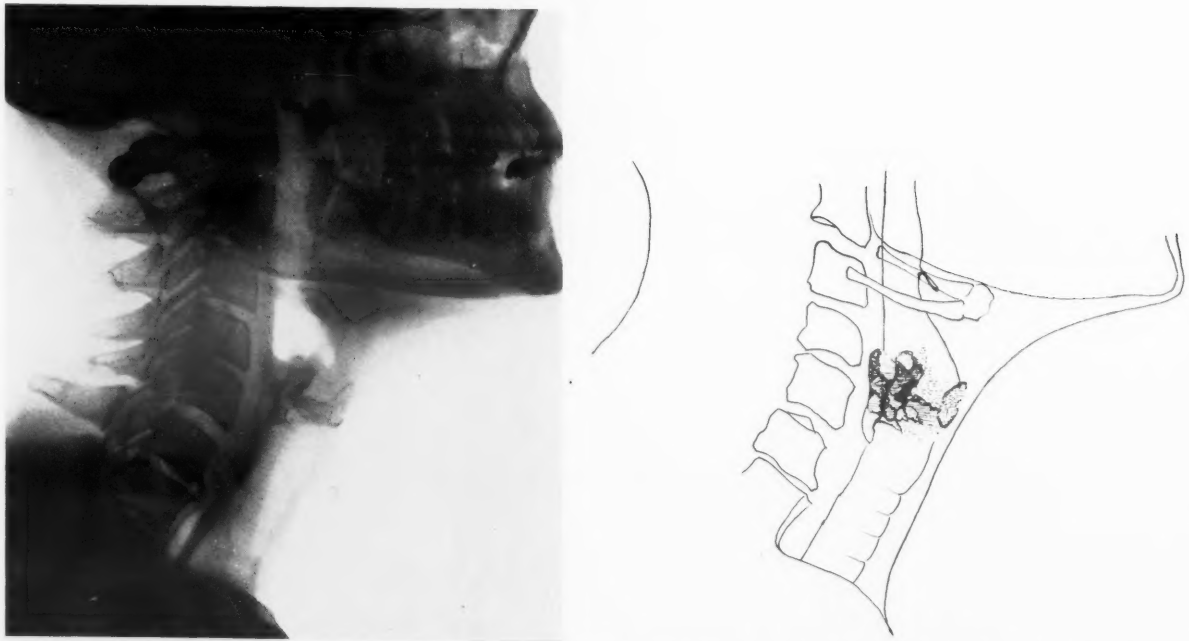
That foreign bodies occurring in the larynx may have been formed there through some pathologic process was suggested more than half a century ago. In the literature reviewed there occur fourteen cases of or associated with ossification of the laryngeal cartilages, and in eight of these bone was expelled. Concomitant factors mentioned were trauma, abscess, syphilitic laryngitis, papilloma and, in the case I reported, carcinoma of the larynx. In one case "a piece of bone" was expelled; in my case, one of the superior cornua of the thyroid cartilage, and in the others the arytenoids, one or both. In

Figs. 6 and 7. Case 3. Roentgenogram and drawing of a larynx of a woman thirty-three years of age showing dense ossification of the arytenoids. This individual is suffering from an advanced case of otosclerosis. The arytenoids are usually the last cartilage to undergo ossification.

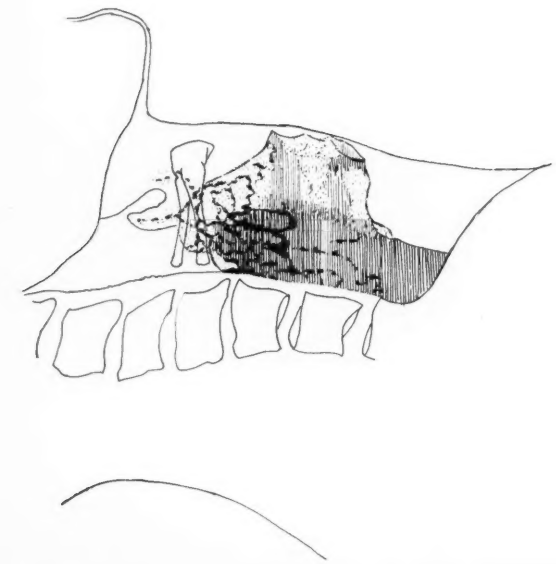
not one of these eight cases was the presence of the foreign body diagnosed; yet in all of them doubtless there was evidence of an inflammatory process, caused by the misplaced bone or sequestrum lying somewhere within the laryngeal tissues. Roentgen studies might have made possible recognition of the foreign bodies. These occurrences indicate the importance of more frequent roentgen studies of morbid conditions of the larynx, especially where there is evidence of change caused by deep inflammatory processes.

In these inflammatory processes in older persons in whom the laryngeal cartilages may have undergone a complete ossification, with the periosteum replacing the perichondrium, roentgen study seems essential. The possibility of inflammatory change, such as an osteitis in the ossified cartilage, should always be considered and an effort made to differentiate this condition from a perichondritis. Perichondritis and chondral necrosis have been mentioned as etiologic factors of abscess of the larynx but misplaced ossified cartilage is also to be considered.

Trauma in relation to ossification of the laryngeal cartilages is of interest. In one case in which the right vocal cord was fixed and the trachea pushed to the left side, roentgenograms showed a fusion of the third and fourth vertebræ and "what appeared to be a foreign body, a quarter of an inch long by one-sixteenth of an inch in width, lying horizontally at about the cricoid in the tracheo-esophageal wall." A history of a minor injury in an automobile accident in which the patient was thrown forward and downward suggested the foreign body might be ossified laryngeal cartilage "thrown off" at the time of the accident and embedded in the larynx, interfering with the movement of the right cord. In another case a man, aged fifty-eight years, who had fallen from "a great height," complained of aphonia and, six months later, expelled the left arytenoid cartilage, completely ossified. In still another case, the patient had been thrown from a horse and had fallen on his head. Ossification of the cartilage in the region of a tracheotomy tube, worn for a long time, has also been mentioned. In a case of injury to the larynx in a man, aged twenty-three years, a roentgenogram taken after the subsidence of the acute chondritis and perichondritis resulting from the trauma, showed a definite acceleration of the process of ossification in the cartilages. The case of a man, aged sixty-eight years, who was suffering from a blow on the larynx, came under my observation recently and suggested the importance of considering ossified cartilage and possible fracture in such a case, for there may be misplaced bone acting as a sequestrum and resulting in abscess of the larynx.



Figs. 8 and 9. Case 4. Roentgenogram and drawing showing bands of ossification throughout the thyroid cartilage. The patient, a woman thirty-five years of age, developed a unilateral abductor paralysis after a direct blow over the larynx.



Figs. 10 and 11. Case 5. Roentgenogram and drawing showing carcinoma of the larynx having invaded surrounding tissues.

Figs. 12 and 13. Case 5. Roentgenogram and drawing three months after roentgenogram therapy. A detached superior cornua of the thyroid cartilage can be observed.

Since sensitiveness of the laryngeal cartilages to radiation is most marked at the site of greatest calcification, the dangers of damage to the larynx from roentgen therapy are apparent. In all severe inflammatory reactions to endolaryngeal burns, whether due to the actual cautery, hot liquids, chemicals or radiation, the possibility of an ossified cartilage lying loose in the larynx is to be considered, particularly when there is evidence of pus formation or a discharging fistula. Too, perhaps such damage may simply hasten the osseous change. Fourteen years ago, a child, aged four years, suffering from papillomatosis of the larynx, came to me in such condition that an emergency tracheotomy was necessary. The multiple papillomata were removed but they returned. The patient was taken elsewhere for treatment, but came back after two years showing evidence of excessive roentgen therapy in the region over the larynx. She has since been under my care. Throughout th_ fourteen years the papillomata have continued to recur and have extended as far down as the bifurcation of the trachea. In this girl, now eighteen years of age, roentgen studies showed a definite ossification of the laryngeal cartilage.

In several cases of advanced otosclerosis, in which the patients were under forty years of age, it has been of interest to me to observe marked ossification of the laryngeal cartilages. The series is as yet not sufficiently large to warrant deductions, but it stimulates further interest and suggests the value of roentgen study of the larynx in all cases of otosclerosis.

Roentgen study of the larynx opens a fertile field of investigation which the roentgen ray alone makes accessible. Close co-operation between the laryngologist and the roentgenologist should make of it a valuable source of information as to the reason and meaning of the process of ossification. Some authorities are of the opinion that in cases of tuberculosis only 50 per cent are recognized by means of the stethoscope, but 95 per cent are diagnosed through roentgen study. Certainly today examination of the sinuses is not complete without roentgenograms. Laryngoscopy, both direct and indirect, is of course indispensable, but in cases of abscess of the larynx or of suspected perichondritis, indeed in every case of laryngeal edema, roentgen study might explain pathologic changes as no other procedure could. The time is rapidly approaching when study of the larynx will be no longer complete without roentgen examination. With improved equipment and technic, the roentgenologist is now able to render invaluable aid in diagnosis, ofttimes disclosing information otherwise available only at autopsy.

Figs. 14 and 15. Case 5. Roentgenogram and drawing three months later of same case after one of the superior cornua had been expelled during a severe paroxysm of coughing.

The wide variability of the age period in ossification of the laryngeal cartilages suggests such causative agents as endocrin disturbance, vitamin or mineral imbalance, improper metabolism or disturbed electrolyte balance which, when understood, will make possible the control of abnormal aspects of this phenomenon. Age, however, remains the primary factor, the process beginning later and advancing more slowly in women than in men. Normally ossification occurs first in the thyroid, next in the cricoid and then in the arytenoid cartilage. Some diseases seem to retard, others to hasten it. In cases diagnosed as perichondritis in the aged roentgen study might determine that the cartilages have been transformed into bone. Also, as an etiologic factor in abscess of the larynx it might disclose an ossified portion of laryngeal cartilage acting as a sequestrum. In fact, roentgen studies of morbid conditions of the larynx, particularly where deep inflammatory processes are present, are of inestimable value in diagnosis and should become an integral part of laryngeal examination.

The writer wishes to express appreciation to Dr. H. B. McEuen for his roentgenologic studies and to Miss Yvonne Kuhne for the pen illustrations.

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XC

BRONCHOSCOPIC ASPECTS OF HEMOPTYSIS*

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NEW YORK

In this paper hemoptysis is understood to mean the expectoration of varying quantities of blood from a lesion somewhere in the lower respiratory tract. The lower tract would include the larynx, trachea, bronchi, the peripheral bronchioles and the pulmonary tissue. The amount of blood may vary from a few drops to a pint or more. The appearance of blood in the sputum is always a cause of grave concern to the individual, his family and his physicians. The family physician is usually the first to be consulted. Most frequently he thinks of bleeding from varices at the tongue base or from the nasopharynx, and refers the patient to a laryngologist. Invariably the laryngologist finds nothing at the tongue base, while the nose and throat are negative for a focus which may have bled recently. I should like to emphasize that bleeding from tongue base varices is extremely rare and therefore should not be considered too seriously when a patient complains of blood spitting. None of the many cases of expectoration of blood which has come to our notice has shown evidence of bleeding from a varix of the tongue base. In the past unnecessary stress has been placed upon this possible source of bleeding. When an individual has spat up blood it would be well to consider that the bleeding came from the lower respiratory tract, unless such an obvious condition as a bleeding tooth socket, a nasal hemorrhage or trauma to the tongue base or the pharynx exists. It would be safe to estimate that more than 95 per cent of small expectorations of blood not associated with vomiting or regurgitation come from the lower respiratory tract. Physicians should not dismiss blood spitting after a routine nose and throat examination which is found to be negative, but should bear in mind the bronchopulmonary tract.

Bronchoscopy has by now established the fact that not every hemoptysis is of tuberculous origin. There was a time, however, when the spitting of blood was at once considered a symptom of tuberculosis. Today the clinician who encounters these cases thinks not only

*From the Department of Otolaryngology, Kings County Hospital.

Presented before the nineteenth annual meeting of the American Bronchoscopic Society, Detroit, May 27, 1936.

of tuberculosis, but also of bronchiectasis, neoplasms, ulcerations, severe passive congestion and several other local bronchial lesions. Credit for the change in the conception of the causative factors of hemoptysis rightly belongs to the enlightening work of the bronchoscopist.

Bleeding from the larynx requires but little discussion. One rarely finds a bleeding point in the larynx in an apparently healthy individual who has recently suffered an hemoptysis. A rare exception to this is the case of telangiectasis of the larynx reported by Zambrini.[9] In such a case the larynx is only one area manifesting the lesion of hereditary multiple hemorrhagic telangiectasis, Rendu-Osler's disease. Bleeding from the larynx may follow the trauma of operation or of external violence, and may also complicate extensive ulcerations, such as can occur with cancer, tuberculosis or syphilis. These ulcerations are usually accompanied by streaking and not by any quantity of blood, as occurs from the bronchus. Hemorrhages of real size, however, are almost always from the lung itself, occurring most frequently from a tuberculous focus which contains an exposed blood vessel.

An understanding of the texture and the condition of the bronchial mucosa may prove of value to the bronchoscopist. During bronchoscopy the lip of the bronchoscope will frequently cause bleeding when it touches the bronchial mucosa, while in many cases it is difficult, ofttimes impossible, to cause bleeding, even deliberately. That different degrees of friability of the small bronchial submucosal vessels exist in different individuals must be apparent to every bronchoscopist of experience. Whether this state of the mucous membrane and its vessels is due to changes in water balance or to vasomotor changes, or to dryness and congestion from local disease, may be very difficult to determine in any given case. In this connection I believe that an increased dryness and hyperemia exists after acute inflammation of the bronchial mucous membrane. One must be careful, therefore, when searching for the cause of hemoptysis, not to mistake the bleeding which may be caused by his bronchoscope for bleeding from the focus which he is seeking.

A condition which strongly merits our attention, because it is frequently written and spoken of, and appears to be nonexistent, is that of varix of the bronchus. Varix, as it is seen in the lower esophagus and elsewhere, does not exist in that portion of the bronchial tree which can be visualized with the bronchoscope. The reason for this is at once apparent when the anatomy of the bronchial

wall is considered. The veins are far removed from the mucosa of the bronchus, being situated at the outermost aspect of the tube. Between the vein and the mucosa there is interposed a firm connective tissue in addition to the bronchial cartilaginous rings. Pathologists are in accord with the writer's contention that varix of the main bronchus does not exist. Tissue changes, both pulmonary and bronchial, are quite frequent and of great variety in chronic tuberculosis, yet one does not encounter varix of the bronchus in these cases either during bronchoscopy or at autopsy. Marked dilatation of bronchial blood vessels is encountered in severe cardiac disease, but this condition does not concern the bronchoscopist.

Tuberculosis takes first rank among the causes of hemorrhage from the lower air passages. Norris and Landis[1] mention an incidence of 30 to 80 per cent, as given by various authors, for this symptom in tuberculosis. In their series of 5,856 patients, 47.6 per cent suffered with hemoptysis. Riessner[5] has recently found that 27 per cent of 5,000 patients with pulmonary tuberculosis had frank hemoptyses.

The question of when to perform bronchoscopy in relation to the time of the hemoptysis has lately received special attention. Some bronchoscopists advocate that this procedure be carried out as soon as possible after the hemoptysis. The writer is not in agreement with this attitude. He feels that only in very small hemoptyses is such a course permissible. It is important, when discussing the question of whether bronchoscopy should be done immediately after the bleeding or not, that the amount of hemorrhage be taken into consideration. Profuse hemoptysis would constitute a contra-indication to bronchoscopy immediately after the bleeding. Very small quantities, or streaking, would not contra-indicate bronchoscopy, regardless of how soon after the hemoptysis one might wish to perform it.

It is well recognized that the spread of tuberculosis and of bronchiectasis to healthy parts of the lung is frequently caused by hemoptysis. If bronchoscopy is done too soon after bleeding, a second hemorrhage may be generated. It is, therefore, important not to bronchoscope patients who have had a recent hemoptysis. Further, there is nothing to be gained from bronchoscopy immediately after hemoptysis. If the causative lesion is localized in the bronchus it can be seen at any time; if it is not localized in the bronchus and is of any consequence, physical examination and x-ray study will suffice to localize it.

Because of the fact that one cannot always be sure of the nature of the cause of the bleeding, and, therefore, cannot exclude tubercu-

losis and bronchiectasis in many cases, it would seem safer to wait a reasonable time, in our experience seven to ten days, before bronchoscopy is performed. As Riessner points out, hemorrhage means acute aspiration, impaired respiration, anxiety, recumbent position, narcotics and their inhibitory effect on respiratory movements, together with suppression of the cough reflex.

Attention should be directed to the fact that anesthesia of the lower air passages, as it is induced when concain solution is dropped into the trachea and bronchi, favors aspiration and a spread of disease. We have seen this occur in both the actively tuberculous and in bronchiectatics. We, therefore, advise caution not only when the sputum is positive, but also when bleeding may be caused by the bronchoscopy.

Perhaps the most manifest causes of hemoptyses are the various neoplasms. Our experiences are in accord with those of Wessler and Rabin,[8] Kernan,[1] Kramer[2] and others that the most constant symptom of benign neoplasms, and particularly the adenomas, is hemorrhage. When one encounters a neoplasm which is covered with mucous membrane and which bleeds readily and profusely, he should suspect that he is dealing with an adenoma. Occasionally, one finds a neoplasm which has all the outward appearances of an adenoma and bleeds just as readily, but which biopsy proves to be a carcinoma.

During expiration, when the lumen of the bronchus is narrowed, the exposed surface of the neoplasm comes into contact with the opposite bronchial wall, and erosion of the new growth takes place. Either a slight ooze or active bleeding may result, depending upon the presence or absence of blood vessels in the eroded area.

It is not the purpose, nor can it be the scope, of this paper to include every possible cause of hemoptysis. Mitral stenosis, chest injuries, aortic aneurysms and infarction are definite clinical conditions whose symptomatology is well understood. These can be readily omitted. It is rather our purpose to discuss such causes of hemoptysis which are little known or understood and which are occasionally referred to the bronchoscopist. In this group might be included such a condition as telangiectasis of the bronchus originally described by Ullman.[6]

There is a large group of cases whose main symptom is hemoptysis in which physical examination and x-ray study yield negative results. This group includes many cases where an ulceratin of the bronchus cannot be detected. Unfortunately, a majority of these

patients are considered tuberculous, even though there is no discernible focus. Yet, as a rule, they are not. Their clinical stories vary: Some patients have only a single hemoptysis and no further symptoms; others have repeated hemoptyses over periods of years, with otherwise excellent health and always absent x-ray and physical signs during the intervals between the blood spitting. Another group expectorate blood, after having had a severe upper respiratory infection. In some a few transient rales are heard at the lung base. The patient coughs for a few days, a week or two, at the end of which time his clinical picture is usually ended. Recently the writer bronchoscoped a man in his late forties, who had been having recurrent hemoptyses for twenty-eight years. Neither x-ray nor physical signs were helpful. When an applicator was passed into the mesial branch of the left lower lobe bronchus, a severe hemorrhage occurred. In this case there very likely existed an exposed blood vessel or a vascular lesion. Another patient had recurrent hemoptyses for seven years. Bronchoscopy revealed the presence of an adenoma of the left main bronchus. A female, twenty-two years of age, suffered recurrent hemoptysis for two years. Her mother had also suffered similarly for several years. The only significant finding at bronchoscopy was the fact that her bronchial mucous membrane bled readily when touched. Whether there was a familial tendency to bleed, as has been reported by Libman and Ottenberg,[3] could not be established. Under the title: "Hereditary Hemoptysis," these authors recorded seven members of a family who suffered with profuse hemoptyses for years, beginning at puberty or early adult life, and not seriously impairing their general health.

In many instances one may be able to elicit a story of exposure to severe cold or to the wind, or to an atmosphere heavily laden with irritating dust. At bronchoscopy one frequently finds a small, superficial ulceration which is trying to heal, usually situated on the floor of the left main bronchus, which, when touched, bleeds. In some cases the ulceration has long since healed and nothing is to be seen during bronchoscopy.

The following cases are illustrative:

REPORT OF CASES

CASE 1.—Mr. T., aged 41, was seen, after he had expectorated blood stained sputum for seven days. This had occurred several times each day. He had been working in a dusty atmosphere for many years, and on the day of first hemoptysis the large loft in which he was employed was being cleaned, so that there was more dust than usual. X-ray study and physical examination were negative. Bronchoscopy revealed an hemorrhagic area upon the inferior wall of the left main bronchus.

When this area was cleansed with a cocain-adrenalin application there was exposed a superficial ulceration, irregularly round and about 3 mm. in diameter. There has been no further bleeding since the bronchoscopy, which was done in 1928.

CASE 2.—On an extremely cold morning Dr. H., aged 47, found the lock of his car door frozen. He proceeded to blow his warm breath upon the lock in order to thaw out the frozen mechanism. About one hour later he began to expectorate small quantities of blood (15 to 20 drops). This continued at intervals during the day and recurred on the day after, so that he sought advice. X-ray study and physical examination were negative. Bronchoscopy revealed a small superficial ulceration on the floor of the left main bronchus. This area was touched with a cocain-adrenalin application. There was no further bleeding.

CASE 3.—Another physician walked in the face of a strong wind on a severely cold day, so that he could feel the cold air rush into his air passages. A few hours later he began to have small hemoptyses. This continued for three days, about two or three times a day. The largest quantity of blood during any one hemoptysis was about one dram. X-ray and physical examination were negative. He was not bronchoscoped, but was kept indoors in a warm atmosphere. His symptoms disappeared within forty-eight hours.

CASE 4.—Mr. U., aged 55, suffered several small hemoptyses during a period of two weeks. Prior to his initial hemoptysis he had had a severe grippe infection. Dr. George Baehr was of the opinion that his previous infection was related to the bleeding. X-ray study and physical signs were negative. He was bronchoscoped five days after his last hemoptysis. Bronchoscopy revealed a healed superficial ulceration of the postero-inferior aspect of the left main bronchial wall. We have seen several similar cases.

CASE 5.—A man, 48 years of age, suffered frequent attacks of hemoptysis. He was first seen in 1928. During the previous fourteen years he had had five attacks. Each time the blood amounted to several ounces in quantity. X-ray study and physical examination were negative. Bronchoscopy revealed a very small quantity of clotted blood in the right main bronchus near the lower lobe branches. He was seen again, some eight months later, because he had just spat up six ounces of blood. He had enjoyed good health during the interval. X-ray study and physical examination were again negative. Several months later he returned because of a similar episode. X-ray and physical examinations were again negative, but this time blood was seen coming from the right upper lobe bronchus. The exact nature of the lesion could not be ascertained.

CASE 6.—A most unusual cause of recurring hemoptysis was seen by me a few years ago. A male, aged 50, was referred because of this symptom. Bronchoscopy revealed a very intense passive congestion of the mucous membrane of both lower lobe bronchi and of the main bronchi just above this area. The mucous membrane in this region appeared to be jelly-like, because so severe a degree of tumefaction existed. The bleeding, which occurred most frequently in the morning and was fairly copious, was no doubt due to contact of the apposing surfaces during expiration, in addition to the stasis which occurred while the patient was at rest during the night. We reported that we saw intense passive congestion and that this must be due to some lesion outside the bronchial tree. The patient was subsequently found to have bilateral calcification of the pleuræ. We elicited the story that he had suffered a severe pneumonia while aboard a slow traveling freighter

some twelve years before. The ship did not carry a physician. Ever since that time he had been short of breath. He had begun to expectorate blood about two years after his illness.

The writer has seen more than thirty patients within the past ten years who suffered with hemoptysis and at the same time had negative x-ray and physical findings. Although a good many of these cases have had superficial mucosal ulcerations, this was by no means the only cause of the hemorrhages. In those cases where an ulceration is found we like to think of the bronchial mucosa as similar to that of the nasal septum, from the standpoint of its vulnerability. Although there is nothing like Kiesselbach's area in the bronchus, one can readily understand how sudden inspiration of excessively cold air or irritating dust can cause abrasion of the mucosal surface, or how severe, acute inflammation may cause local changes in the bronchial mucous membrane. The left main bronchus, being more horizontally placed than the right, should, from the mechanical viewpoint, be more vulnerable. This is borne out by the fact that practically all of the ulcerations seen by the writer have been in the left main bronchus. Wessler and Jaches[7] believe that scleroses of small vessels may be a factor in these unexplained hemoptyses. Hemoptyses such as we are discussing are frequently seen in women. Some feel that the menstrual cycle may have some bearing upon them. Many believe that local vasomotor changes can be sufficiently severe to cause bleeding.

CONCLUSIONS

The general subject of hemoptysis, as it relates to the bronchoscopist, is reviewed. Bleeding from varix of the tongue base should not be seriously considered. It rarely occurs. There exists a relatively large group of cases in which hemoptysis is the predominant symptom, where the patients are in apparently perfect health and where the x-ray study and physical examinations yield negative results. Many of these cases can be found to have superficial ulcerations of the bronchus; in others it is impossible to ascertain the exact cause of the bleeding. Neither anatomic and pathologic studies nor clinical experience justify the impression that varix of the bronchus exists as a cause of hemoptysis. Bronchoscopy might be permissible immediately after hemoptyses which consist of a few drops or a mere streak of blood, but is dangerous, in the writer's opinion, when the bleeding is copious.

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XCI

DIFFICULTIES AND PITFALLS IN THE INSINUATION OF THE ESOPHAGOSCOPE

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The dangers of esophagoscopy are closely interwoven with the difficulties; both will be briefly considered here. Complications will be considered only in their prophylactic phases. This paper is written in the hope of elimination of avoidable mortality.

The difficulties and dangers of esophagoscopy may be classified as educational, anatomic, physiologic and pathologic.

EDUCATIONAL DIFFICULTIES AND DANGERS

It is an unquestionable fact that, whereas any medical graduate can be quickly taught to introduce the bronchoscope safely, teaching the beginner to introduce the esophagoscope is fraught with graver responsibilities than the teaching of anything else in the whole realm of surgery. Elsewhere the misdirected hand or instrument can be checked by the teacher; the esophagoscopic tube mouth can overstretch and split the hypopharyngeal wall before the teacher can discern the misdirected pressure. The first thing to teach the pupil is that if he puts an esophagoscope into a patient's throat and pushes on it the one place it will not go is into the esophagus. Fear of mortality must be instilled until it is a habit of mind. The next thing to teach him is that the slightest trauma to the esophageal wall will be fatal. The necessity for every minute technical detail must be taught. He must be particularly cautioned not to try new methods or short cuts until he has thoroughly mastered the old tested methods. With these precautions inculcated we may then teach the beginner details of technic.

ANATOMIC AND PHYSIOLOGIC DIFFICULTIES

Normal anatomic and physiologic difficulties and dangers, as to the particular patient, are general and local.

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*Presented before the nineteenth annual meeting of the American Bronchoscopic Society, Detroit, May 27, 1936.

General Anatomic Difficulties.—The difficulties and dangers are supposed to be greater, other things being equal, in the powerful, muscular type of patient with a full set of teeth, narrow alveolar arches, with practically no visible neck; his head is set on his shoulders. Yet with proper technic and a well-trained team this type of individual presents no great difficulty and no danger.

Having passed the cricropharyngeal pinchcock the next local anatomic and physiologic difficulty will be encountered at the diaphragmatic pinchcock. Here again, if there is no pathology, the difficulty is not insurmountable and there need be no danger.

Local Anatomic and Physiologic Difficulties.—1. The hypopharyngeal walls are very thin. 2. The cricopharyngeus and its associated muscles are very powerful; they pull strongly on the cricoid and the median raphé to resist the entrance of the esophagoscope. The esophagoscopist must be prepared to deal with these difficulties without danger to his patient.

Personal Equation of the Patient.—The variable anatomic equation has been referred to above. Additionally, there is the temperamental factor in the equation. Some persons have an inordinate terror of being hurt; many individuals are opposed to anything that may involve discomfort. Others have ruined all their good sense and self control by the use of alcohol or tobacco, or both. The present enormously increased use of alcohol will soon develop many of the recently rare, old type of drunkard, who in the old days was well known as a bad subject for any procedure, even an anesthetic, general or local. All these different types are unduly apprehensive and at the start of esophagoscopy their cervical muscles are in an almost tetanic state of contraction. There is a way of dealing with this condition also.

Pathologic Factors.—Disease may weaken the wall of the esophagus; various conditions may thicken it in one portion, rendering more dangerous the normal portions; or they may deviate or obliterate its lumen. All of these conditions add no risk to the manipulations of him who has mastered himself and his esophagoscope.

HOW ARE THE DIFFICULTIES TO BE SURMOUNTED, THE DANGERS TO BE AVOIDED?

Two elements enter into prophylaxis of disaster: 1, the mental attitude of the operator and, 2, his technic.

Attitude of the Operator.—This is of the utmost importance for safety; in fact, it may make all the difference between a surpass-

ing success and a fatal failure. Disaster is born of overconfidence. A great surgeon sent a nurse to borrow an esophagoscope. The patient died promptly and the great surgeon has long since followed the patient to the great beyond. Mosher has said that when an esophagoscope is started down the esophagus there is tragedy in the offing. It is true that this tragedy can be entirely avoided by practice, but it is equally true that the mental attitude implied should never be overcome. The operator should always have uppermost in his mind during the insinuation of the esophagoscope the thought "What if I should cause a perforation?" This thought will make him, with practice, a perfectly safe esophagoscopist.

There is an additional element in the mental attitude that is important, namely, patience. The esophagoscopist who aspires to safety, and, of course, all esophagoscopists do, should develop patience to the highest possible degree. In the esophagus, as at intersections of streets, accidents will result from impatience. It is necessary for the esophagoscopist to wait at the cricopharyngeal pinchcock until he gets the safety signal, which is the relaxation of the cricopharyngeal pinchcock.

As a contribution to a prophylactic conception, the word insinuate is better than introduce, since it implies to introduce slowly, as through a winding and narrow passage. An introduction may be brutal.

Temperamental Equation.—The best remedy for difficulties due to the temperatmental equation of the patient is the sermon on relaxation. This applies also to the pathologic temperament of him who drinks alcoholic beverages. A sedative, given an hour beforehand, will be a helpful adjunct to the sermon in both classes of case.

A Trained Team.—It ought not to be necessary to repeat today what we advocated many years ago. And yet there still appear in the medical journals tables, instruments and devices advocated because it is supposed they will dispense with trained assistants. If training is deemed proper and necessary for winning a football game, is it not equally so to prevent a death from esophagoscopy?

Find the lumen is the best means of overcoming difficulties and avoiding dangers. The esophagoscope should never be advanced until a lumen is seen ahead.

The Lumen Finder.—The best and safest means we have ever found for finding a lumen is the small sized, blunt pointed soft rubber instrument shown in the illustration. Practically it is harmless

if used gently; we have used it for a number of years and have come to have perfect confidence in its helpfulness and safety. When no lumen is visible a search for a lumen is made by gentle palpation with the lumen finder. When the lumen is found the esophagoscope may be gently and safely advanced. The lumen finder is not, in any sense, a mandrin for blind introduction. It is small in diameter; it is passed by sight. When it has found and entered deeply into the lumen, the esophagoscope is advanced while the endoscopist is sighting alongside the finder and watching the folds as they unfold and recede. If they do not unfold no great pressure on the esophagoscope is made until the reason why is determind. Gentle pressure at the right place is all that is necessary; but to gauge gentle pressure it is necessary to eliminate binding of the tube at three points: 1, the upper teeth; 2, the lower teeth, and, 3, the inside of the mandible. The lumen finder is equally useful at the hiatal and the cricopharyngeal pinchcocks. In the latter location it often helps by causing a swallowing movement. Normal deglutition involves the coordinate

Fig. 1. Lumen finder.

opening of the cricopharyngeal pinchcock as the inferior constrictor of the pharynx contracts. When we are at the threshold and our way is barred by a cricopharyngeus that stubbornly delays relaxation, we are often helped to overcome the stubbornness by a swallowing movement resulting from contact of the lumen finder. Requesting the patient to swallow may help.

The foregoing refers to the normal esophagus. The lumen finder is equally advantageous in the various forms of congenital and pathologic stenosis. In these conditions roentgenograms and the fluoroscopic screen are sometimes helpful; for this reason the lumen finder is rendered intensely opaque by a filling of bismuth subnitrate.

SUMMARY

1. Esophagoscopy, considered apart from the condition for which it is done, can be rendered a perfectly safe procedure by careful study, patience, practice, the sermon on relaxation, proper position of the patient, trained assistants, passage by sight, and the use of the lumen finder.

2. Find the lumen is the fundamental rule of safety. No pressure on the lumen is safe unless a lumen is seen ahead or assured by the lumen finder; even then the pressure should be gentle.

3. Insinuation gives a safer concept than introduction as applied to esophagoscopy.

4. Disaster is born of impatience or overconfidence.

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XCII

PULMONARY HEMORRHAGE STUDIED AND TREATED BY THE BRONCHOSCOPIST*

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The bronchoscopist not infrequently is asked to s[illegible]rients with hemoptysis in an effort to establish the cause of ble[illegible]g and to aid in establishing the most satisfactory means of treatment. In studying such cases it is necessary to differentiate between true hemoptysis and bleeding from the pharynx, nose, mouth, etc. Since it usually is possible to find the source of bleeding only when it is active, the necessity for examination at this time is obvious. Occasionally the hemorrhage may be quite severe, and under such conditions we find it is important to be prepared, before beginning the bronchoscopic examination, for immediate transfusion. Such preparation means that a satisfactory donor has been secured and is present in the operating room for immediate use if required. When the bleeding point is located in the trachea or the larger tubes, it usually can be visualized. When it is in the periphery, this is impossible, but by bronchoscopic study, especially when based on information obtained beforehand through physical and x-ray study, localization to a definite area usually is possible. This is important from the standpoint of etiology as well as therapy. Team work between the bronchoscopist, the internist, especially trained in thoracic diseases, the thoracic surgeon in the study of such cases, is of paramount importance in obtaining the most accurate diagnosis and in working out the best treatment.

The causes of pulmonary hemorrhage, in the order of their frequency, are as follows:

1. Tuberculosis (See Case 1, G. C.).

2. Ulcer, frequently associated with bronchiectasis or abscess. Removal of purulent secretions and blood at the time of bronchoscopy helps to avoid "drowning" of otherwise healthy tubes and immediate interference with respiration and possible atelectasis and

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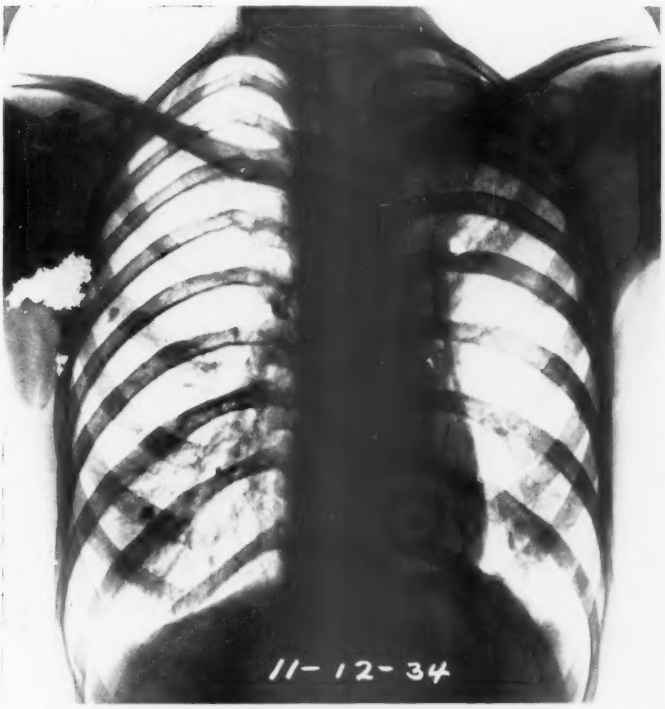

Fig. 1. Case 1. Shows the typical chest which would be expected in an individual with her history. For our purposes atelectasis of the upper portion of the left upper lobe is seen.

infection. Cutting away granulation tissue for improved drainage distal to its level or securing biopsy specimens sometimes is accompanied by rather active bleeding. As a rule, this may be controlled by the application of cocain, adrenalin and pressure for a short time. Occasionally it may be necessary to leave a tampon in position, and these should always be on hand and ready for immediate use. A tampon should never be allowed to remain in a bronchus longer than twelve to twenty-four hours, because of the danger of pneumonia, as a result of interference with lung drainage (See Case 2, M. G.). When a tampon is left in a bronchus it is imperative that it be anchored with heavy braided silk which is long enough to be brought out the corner of the mouth and secured to the cheek with adhesive tape. Patients should never be left alone while the tampon is in position. Those in attendance must be instructed to pull out the braided silk and tampon should it be coughed up into the trachea

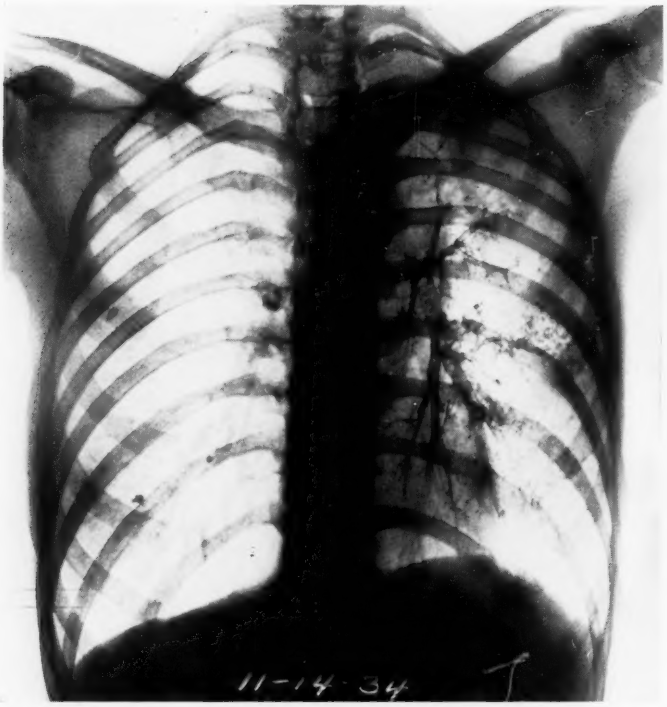

Fig. 2. Case 1. Bronchography shows obstruction of the branch bronchus leading to the apex of the left upper lobe.

or larynx, thus avoiding asphyxiation. On one occasion a nurse saved a patient from such an accident by this procedure.

3. Tumor, most often malignant and less frequently benign. Malignant tumor often is accompanied by abscess formation and ulceration, with erosion of the vessel walls and rupture, as a result of strenuous coughing. Endobronchial tumor, by pressure erosion, may destroy the bronchial wall and that of adjacent vessels, with severe bleeding into a lumen of the bronchus (See Case 3, C. B.).

4. Aneurysm has been seen with erosion into a bronchus and severe bleeding. Foreign body in the esophagus over a prolonged period by erosion due to pressure and infection may destroy the wall of the tracheobronchial tree when severe bleeding is likely to ensue; or an unsuspected foreign body in a bronchus, in addition to causing bronchiectasis, may cause erosion and bleeding (See Case 4, B. C.).

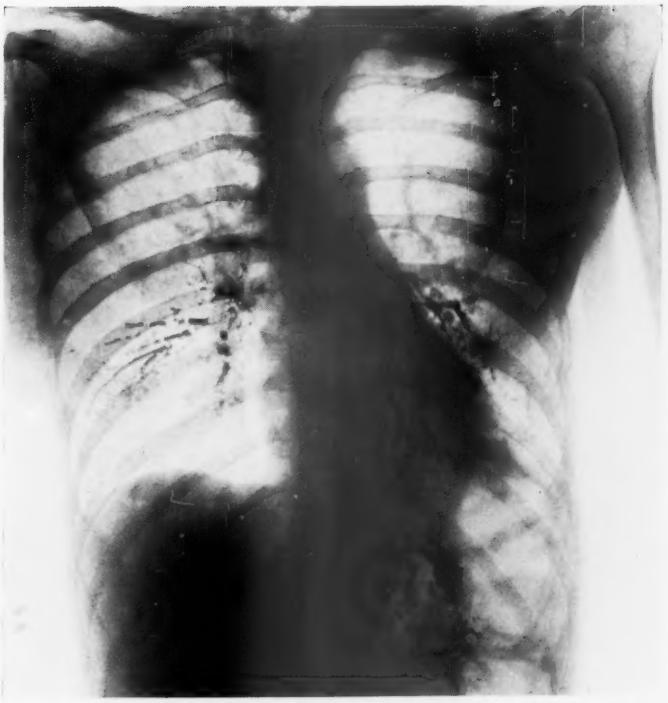

Fig. 3. Case 2. Bronchography shows bronchiectasis of the left lower lobe.

5. Broncholith, impacted or movable, with ulceration and bleeding.

6. Unknown causes, a few cases have been seen in my practice and in the Medical and Surgical Chest Service of the Washington University Hospital Group, in which, after most exhaustive study, the cause of bleeding was not established. These were cases in which the lesion was in the parenchyma. Some of these were undoubtedly caused by a spirochetosis, and in one, syphilis acquired by a transfusion may have been a factor. In none of these cases could we prove the presence of tuberculosis.

In cases of obscure or unknown origin studied in our service, bronchiectasis with ulceration has been the cause of hemorrhage in most of the cases. (See Case 2, M. G., and Case 4, B. C.). Severe bleeding was seen in a case of endobronchial carcinoma, which by pressure erosion had destroyed the wall of the bronchial artery. (See Case 3, C. B.).

Fig. 4. Case 3. Shows a circular mass in the lower portion of the thoracic cavity. X-ray diagnosis of tumor was made.

As a result of the great advances in the surgical treatment of bronchiectasis, renewed interest has been awakened in the study of these cases. Every case of bronchiectasis should have at least one bronchoscopic examination. This is important because not infrequently an unsuspected foreign body is found, and because of the valuable information thus to be obtained in localization of the affected lung. Tuberculous individuals, whose diagnosis has been proven, occasionally bleed from other causes. It is now well known that such individuals may be safely bronchoscoped provided proper care is observed. Here again, the importance and value of team work is emphasized. Occasionally it is possible to prove the diagnosis of tuberculosis only by study of specimens of sputum obtained bronchoscopically (See Case 1, Plates Nos. 1 and 2, G. C.).

Localization of pulmonary bleeding by bronchoscopic examination affords information of the greatest importance to the thoracic

Fig. 5. Case 3. Bronchography shows obstruction of the main bronchus. Diagnosis of tumor was made on this plate.

surgeon when he plans surgical intervention. I find it advantageous to use avertin in patients to be studied bronchoscopically, especially those likely to bleed. This drug usually lowers the blood pressure and nearly always abolishes all feeling of apprehension in the patient and in addition it prevents his having an unpleasant recollection of the procedure. This feature is important because of the likelihood of the need for further surgical procedure of one kind or another at some future date.

REPORT OF CASES

CASE 1.—G. C., white female, aged 34, admitted to Barnes Hospital, November 13, 1934, on the Chest Service. Chief complaints were coughing dyspnea and pain in left chest following a severe cold two months before.

In 1925 the patient had a kidney disorder. The question of tuberculosis as well as hydronephrosis was considered at that time, but not proven. She also had a left pleurisy at that time, and chest plates were suggestive of pulmonary tuber-

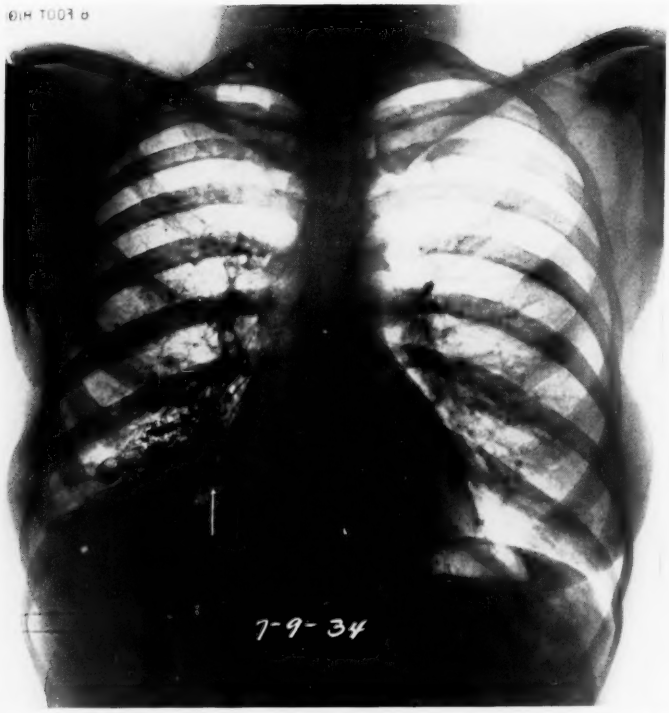

Fig. 6. Case 4. Bronchography shows bronchiectasis of the right middle and lower lobe. On careful study a shadow of a tooth will be noted which is somewhat blurred by the lipiodol.

culosis. No tubercle bacilli were found. Family history shows that the father died of tuberculosis.

Physical examination revealed a small area of atelectasis in the extreme apex of the left upper lobe (Fig. 1). The x-ray findings, both by plate and fluoroscopic examination, were typical of this condition. Bronchography revealed blocking of the first division of the left upper lobe (Fig. 2). Bronchoscopic examination revealed an ulcer in the bronchus to the left upper lobe at the level of its first division. In appearance this ulcer was highly suggestive of tuberculosis. Smears from the bed of the ulcer showed many tubercle bacilli, and guinea pig inoculation was positive. We recovered a moderate amount of blood-streaked pus and probably removed the obstruction. This specimen contained a large number of tubercle bacilli. With appropriate treatment the patient made a prompt recovery, and within six months returned to part time work and is now on full time duty.

Case 2.—M. G., white female, age 28, admitted to Barnes Hospital, June 23, 1932, with persistent hemoptysis. She was known to have bronchiectasis, and a bronchoscopy was requested in order to localize, if possible, the bleeding point.

The tracheobronchial tree contained large quantities of foul pus. During the process of removing this with suction, a very free hemorrhage came from the left main bronchus, very probably the result of injury to a blood vessel with a weak spot in its wall. The bleeding was controlled only by the use of tampons. These were left in position two days. The patient developed a fatal pneumonia.

CASE 3.—C. B., white female, age 44, admitted to Barnes Hospital on August 9, 1931, with a history of cough, hemoptysis, and loss of weight. She had had a similar attack twenty-four years previously but had apparently recovered from that attack.

In 1930, she had an attack accompanied by a mild fever, severe paroxysms of coughing, with tenacious blood-streaked sputum. She lost five pounds in the year prior to admittance, and had had dull aching pains in the right chest.

On admission the plates showed a tumor (Fig. 4), which was thought to be possibly in the posterior mediastinum, and that the sputum might be due to passive congestion. Lipiodol plates (Fig. 5) showed a definite bronchial obstruction, apparently caused by a mass within the lumen. Bronchoscopy revealed a tumor in the right main bronchus which filled the lumen but was easily movable. A biopsy specimen was diagnosed as nonmalignant tumor. There was no bleeding at this time.

Within a few days preparation was made to remove the tumor through the bronchoscope under avertin and local anesthesia. Cocain and adrenalin were gently swabbed on the surface of the tumor and on the surrounding bronchial wall. As this was done the position of the tumor was shifted somewhat, and while waiting for the local anesthesia to become effective there occurred spontaneously a very severe hemorrhage. The bleeding apparently was from a large artery. Its volume was sufficient to fill a 9 mm. bronchoscope, and it spurted freely from the bronchoscope. It was necessary to use tampons to control the bleeding. Four tampons were required. These were removed on the following morning without renewed bleeding.

The patient was referred back to the thoracic surgeon for treatment by external operation. She was finally obliged to accept external operation by reason of the increasing severity and frequency of the hemorrhage. When the chest was opened and the lung picked up, the pleural cavity immediately filled with blood. This was stopped with difficulty, and the patient succumbed within the hour in spite of all supportive measures.

On postmortem examination we found that as the result of pressure erosion there was a considerable opening into the bronchial artery which communicated directly with the lumen of the bronchus when the tumor was raised from its bed. So long as it was allowed to remain in its original position it seemed to block the opening into the vessel almost completely, except when coughing.

CASE 4.—B. C., colored, admitted to Barnes Hospital, June 19, 1934. Chief complaint was coughing and hemoptysis. The past history was negative.

There was evidence of bronchiectasis in the right lower chest, involving the middle and lower lobe (Fig. 6). In one plate was seen an object which resembled a tooth. Bronchoscopic study, aided by fluoroscopic guidance, enabled us to pick up a tooth which was distal to a cicatricial stenosis of the bronchus of the middle lobe and rested in a large cavity. Because of the bronchiectasis and the extremely narrow stricture it was felt better to refer the patient for external surgery in order that the bronchiectasis might be dealt with at that time. There was free bleeding during the time.

539 NORTH GRAND AVENUE.

XCIII

SOME PROBLEMS IN ESOPHAGEAL ATRESIA

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The paper will present a discussion of two important problems in esophageal atresia: first, the impermeable stricture, and second, the congenital atresia with tracheo-esophageal fistula.

A review of the literature indicates that true impermeable stricture of the esophagus is uncommonly encountered. As a rule, a small passageway persists which permits string swallowing with subsequent safe dilatations, preferably by the retrograde technic. Atresia may be considered complete when in the presence of a gastrostomy fistula no saliva can be found nor ingested dyes, such as methylene blue, detected in the stomach contents. Various procedures have been tried successfully in reported cases: ballooning of the esophagus from above and picking the stricture apart by means of a small metal olive or forceps (Mosher[1] and King[2]); combined peroral and retrograde esophagoscopy under double plane fluoroscopic guidance (Jackson[3]); the use of electrolysis (Guisez[4] and Heindl); retrograde bouginage with the aid of a cystoscope inserted through a gastrostomy (Iglauer[5]); and peroral esophagoscopy combined with digital guidance and instrumentation from below (Brenneman, Jackson,[6] Wishart[7] and Briggs) or from above (Imperatori[8]).

I wish to report a case of complete atresia successfully treated by peroral esophagoscopy combined with digital and instrumental approach through an opening in the stomach.

REPORT OF A CASE

CASE 1.—S. C., a man of 47, was admitted to the Strong Memorial Hospital on April 4, 1930, complaining of inability to swallow, with an accompanying loss of about twenty pounds in weight. He gave a history of intermittent dysphagia over the preceding eight years. During this period he had been treated by his medical adviser with antispasmodics and occasional dilatations with a soft bougie. X-ray study some time during this period was said to have demonstrated a typical cardiospasm. He worked in the chemical division of a large industrial plant but had never to his knowledge ingested any caustic. His present attack had begun twelve days before and had not responded to medical treatment. The physical examination

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showed nothing of interest except the evident loss of weight. There was a moderate secondary anemia. The Wassermann reaction was negative throughout. An x-ray study showed considerable dilatation of the esophagus above what was considered to be a typical cardiospasm. A direct esophagoscopy was performed and confirmed this impression. Considerable gluing together of the vertical folds below the diaphragmatic level was noted. The stomach was entered with a 9 mm. scope and the terminal esophagus dilated. Immediate improvement in swallowing was noted. The patient was advised to report back in two weeks but did not do so and was not seen again until April 16, 1932, two years later, when he returned to the hospital complaining of complete inability to swallow. His symptoms of obstruction had returned three months before and had grown steadily worse. He had lost about forty pounds in weight and was very weak. X-ray studies (Fig. 1) showed complete obstruction at the level of the diaphragm and fluoroscopic rechecks several hours later showed no evidence of the barium having entered the stomach. A direct esophagoscopy was done and a complete obstruction encountered 39 cms. from the upper incisors. It was impossible to coax the smallest filiform bougie through this obstruction nor could any point of continuation be detected after ballooning the esophagus. Immediate gastrostomy was considered indicated and was done on April 18 by Dr. S. J. Stabins. A high caloric liquid diet was instituted through the tube and rapid improvement in the general condition followed. Several attempts were made to swallow a thread but were entirely unsuccessful. Two weeks later, May 1, a retrograde esophagoscopy was done. The cardiac orifice of the esophagus could be readily identified, but neither bougie nor ureteral catheter could be passed up into the esophagus. The diagnosis of complete atresia was further confirmed by the absence of saliva in the gastric contents as well as by the fact that methylene blue given by mouth failed to appear in the stomach contents. On May 2, under ether anesthesia, the gastrostomy incision was enlarged upwards and the sinus tract excised. The stomach opening was continued high enough on the anterior wall to permit direct inspection of the cardiac orifice. Digital examination encountered complete obstruction just above this point. There was nothing to suggest malignancy. Efforts to introduce a ureteral catheter were also entirely unsuccessful. At this stage a 9 mm. esophagoscope was passed to the point of obstruction and its tip could be definitely palpated by the surgeon's finger. The thicker walls as well as the more axial approach from below, in our judgment, rendered penetration of the stricture safer from below than from above. For this reason a small metal dilator was gently forced up through the stricture until it appeared in the esophagoscope. A ureteral catheter was then introduced and drawn up into the mouth and a continuous string of heavy braided silk effected. Following this procedure the patient ran a low grade febrile course ranging from 37° C. to 38° C. for five days. On the first postoperative day the W. B. C. was 26,000. On the seventh postoperative day, May 9th, retrograde dilatations were begun, using a No. 12 Fr. Tucker bougie. The first dilatation was followed by considerable pain in the right shoulder and by a sharp elevation of temperature to 39° C. The pain lasted for about four days and was accompanied by cough productive of small amounts of purulent sputum. An x-ray study at this time showed no definite evidence of mediastinitis or lung changes. After two days of normal temperature dilatations were resumed, this time without reaction. Increasing size bougies were used at two-day intervals and retained in the area of stricture for twenty-minute periods. The stricture opened rapidly, and by May 20th, the eighteenth day postoperatively, he was taking all his feedings by mouth. The patient was discharged from the hospital on June 11th. Retrograde dilatations were continued

at increasing intervals until July 1st, two months after operation, at which time the continuous string was removed. At this time a 7 mm. full lumen scope could be easily passed into the stomach. The gastrostomy opening was kept patent for ten months and then allowed to close. The patient has been followed since that time and direct examinations done three or four times each year. He has not developed any recurrence of symptoms, although when he is nervous or upset swallowing may be temporarily difficult.

Comment.—This case of complete atresia of the lower end of the esophagus is of interest because of the preceding history of cardiospasm, confirmed by roentgenologic as well as direct endoscopic studies two years before. It illustrates one method of management of this hazardous condition. Mosher[9] has emphasized the frequency with which the esophagus may become infected in the course of acute or chronic diseases, by inflammation spreading from chronic infections of neighboring organs, as well as from within by way of the glands. It is of interest in this case to recall his opinion that the essential lesion in cardiospasm is a fibrosis of the esophagus in the region of the crural canal in association with a twisting of the terminal portion of the esophagus. In the management of atresia of the esophagus, regardless of the method of treatment employed, it is, in our opinion, of the utmost importance that the gastrostomy opening be maintained for a full ten or twelve months and that such patients be followed for a long period with occasional direct endoscopic studies in order to detect and prevent a possible recurrence.

Congenital atresia of the esophagus has been a subject of interest and speculation to clinicians for many years. Originally entirely a curiosity, the gradually increasing number of reported cases convinces one that the condition is actually more common than was formerly recognized. Plass,[10] in 1919, was able to collect from the literature 136 verified cases of this anomaly. Rosenthal,[11] in 1931, assembled 85 additional cases. Since then many other reports have been published, including interesting presentations before this Society by Imperatori[12] and Iglauer.[5, 13]

These reports have served to clarify our ideas concerning the clinical picture as well as the diagnostic criteria of this condition. The treatment, however, still remains as undetermined as ever, and as yet no case of successful management has been reported. This problem therefore must continue to draw our attention until some practical method of management has been evolved.

As a result of a study of reported cases certain facts concerning the pathologic and clinical picture emerge as generally accepted.

Pathologic Data.—In the common type of this anomaly which, according to most observers, constitutes over 70 per cent of cases,

the upper segment of the esophagus begins normally but ends as a blind sac within the thoracic cavity at varying distances below the larynx. Its average length is about 3.4 cm. (Plass). The pouch is generally hypertrophied and uniformly dilated to a diameter of about twice that of the normal esophagus of the newborn. The lower segment is entirely separated from the upper one and makes a fistulous connection with the trachea in the region of the bifurcation, the usual point being about 0.5 cm. above it. Occasionally the opening is into a main bronchus near the carina. The muscular walls of the esophagus and trachea blend without any demarcation. The esophagus at this point is very narrow, being about 3 mm. in diameter for a distance of 4 to 7 cm., and then gradually increases to its normal caliber as it approaches the stomach. Although occasional overlapping of the two esophageal segments has been recorded (Keith,[14] Rosenthal), usually they are separated by a distance of about 1 cm., and in some cases by as much as 4 or 5 cms. The two segments are frequently bridged by a dense fibromuscular cord. Other congenital anomalies are commonly associated with this condition. In Plass' series they were present in 59 of 94 cases. Few of them, however, were incompatible with life, the most common being atresia ani.

Clinical Data.—The clinical picture in reported cases is strikingly uniform. Typical signs are noticed as soon as feedings are begun. After a few swallows fluid is returned through the mouth and nostrils. This is followed by cyanosis, struggling, coughing and frequently by brief periods of unconsciousness, during which artificial respirations may be necessary. Drowning appears imminent, but is avoided, as a rule, by drainage of the fluid from the trachea into the stomach by way of the tracheo-esophageal fistula. The weight falls rapidly and evidences of marked dehydration appear early. Death from starvation and bronchopneumonia occurs in most cases within the first two weeks.

The important diagnostic criteria, in addition to the clinical history, are: First, distension of the upper part of the epigastrium with tympany on percussion due to the fistulous communication between the stomach and lungs. In such cases the roentgenograms show air in the stomach, which is sufficient in itself to establish the diagnosis of a tracheo-esophageal fistula. Second, the blind upper pouch can be identified under the fluoroscope and in the roentgenogram by the passage of an opaque catheter and outlined by opaque media, preferably lipiodol. It must be emphasized that barium should be avoided. In one of our cases subsequently reported scattered minute lung abscesses were observed at autopsy about particles of aspirated

barium. The upper pouch may also be studied by direct esophagoscopy, and as Tucker and Pendergrass[15] have demonstrated, the fistulous opening may be identified through the bronchoscope.

Prognosis and Treatment.—Treated or untreated, this condition has proved universally fatal. Most infants have died within the first two weeks. Phelps[16] reports a case living eighteen days. Beattie,[17] in discussion and personal communication, describes a case treated only by gastrostomy, which survived for 144 days. It is probable in such a case that the communication with the trachea was unusually narrow so that fatal regurgitation was limited for a considerable period. Certainly universal experience as well as understanding of the underlying pathology has demonstrated the futility of gastrostomy and jejunostomy. Various transthoracic procedures have been proposed. Richter,[18] in 1913, attempted closure of the lower segment at its junction with the trachea, followed by gastrostomy, but both his patients died shortly after operation. No other cases in which this procedure has been tried have been reported. Iglauer,[13] in 1934, proposed a two stage operation consisting of "cervical esophagostomy for drainage of the blind esophageal pouch," followed by a "transthoracic operation for externalization of the lower esophagus, i. e., dorsal esophagostomy for feeding purposes." No actual clinical use of this procedure has as yet been reported. Smith[19] and Ravdin[15] have tried gastrostomy combined with tying off the lower end of the esophagus below the diaphragm. Smith's case died twenty hours after operation. Sarnoff,[20] in addition, transected the lower end of the esophagus and brought the stump to the surface as an abdominal esophagostomy. His patient succumbed on the thirteenth day of an aspiration pneumonia.*

*Just prior to this meeting an article by Gage and Ochsner (Gage, M., and Ochsner, A.: The Surgical Treatment of Congenital Tracheo-esophageal Fistula in the New-Born. Ann. Surg., 103:725, 1936) has appeared recommending intra-abdominal ligation of the cardiac end of the esophagus with umbilical tape and the formation of a gastrostomy of the Ssabanejew-Frank type. They report two cases, one dying on the third and the other on the twenty-seventh day. In the second case, operation was not done until the twenty-fifth day. Mixter, in discussion, describes an approach to the site of the fistula through the posterior mediastinum on the right side. The lower segment is severed from the trachea and implanted in the skin incision. Feedings are conducted through a catheter placed through the esophagus into the stomach. Proximal esophagostomy is done at once or within a short period. In two out of nine cases the infants have survived for fifteen and thirty days respectively.

REPORT OF CASES

I wish at this point to report two cases which are of particular interest because of the length of time which they survived.

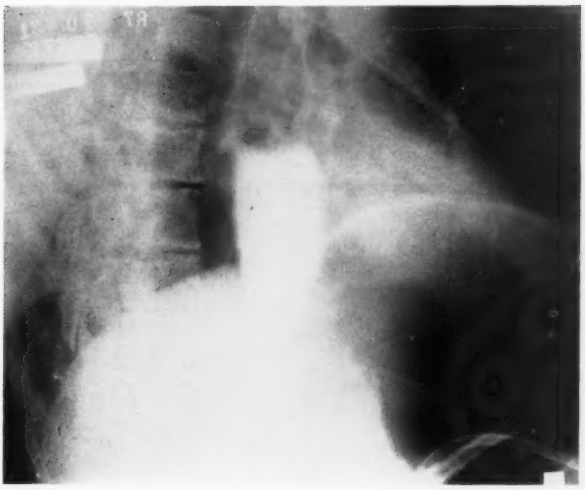

Fig. 1.

CASE 1.—J. M., a male infant, was admitted to the Strong Memorial Hospital on August 4, 1928, five days after birth. The child had been full term and the delivery spontaneous. Considerable drooling of saliva had been noticed from the first day and when nursed typical choking spells with coughing, cyanosis and regurgitation were observed. Barium studies had already been made at another hospital and a blind upper esophageal pouch outlined. The upper part of the epigastrium was tympanitic on percussion and the roentgenogram showed considerable air in the stomach. We concurred therefore with the admission diagnosis of congenital esophageal atresia with tracheo-esophageal fistula. The infant was given 130 cc. of saline intraperitoneally and 150 cc. of glucose subcutaneously in two divided doses at four hour intervals. A laparotomy was then performed by Dr. W. J. M. Scott. The upper end of the stomach was transected. The distal end was carefully inverted with a double row of silk sutures, completely peritonealizing the wound. The proximal portion was brought up to the abdominal wall to form an abdominal esophagostomy. A gastrostomy opening was then established. The patient withstood the operation nicely and made satisfactory progress until the thirtieth day when signs of bronchopneumonia became evident. Death occurred on September 12th, thirty-four days after birth. During the postoperative period secretions from the abdominal esophagostomy were irritating and troublesome.

Autopsy Findings.—The esophagus (Fig. 2) was found to consist of a blind dilated upper segment, still containing considerable barium, and ending 3.5 cm. below the larynx. The lower segment entered the trachea 1.5 cm. above the bifurcation. The two segments were separated by a distance of 1 cm. and were bridged by a thick band of dense connective tissue. The tracheal end of the lower segment measured about 3 mm. in diameter for a distance of 5 cm., after which it rapidly reached a normal caliber. The lungs showed evidences of a bronchitis and a diffuse bronchopneumonia. Scattered through the right lung were small discrete greenish yellow areas which on microscopic study were found to be small abscesses occurring

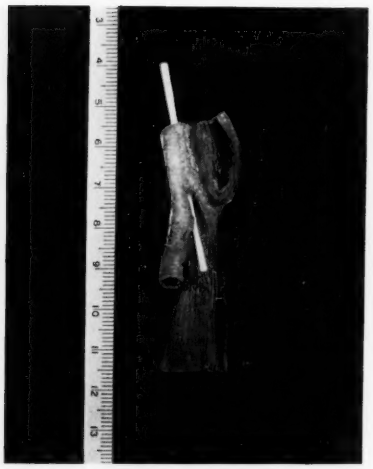

Fig. 2.

around particles of aspirated barium. There were no signs of other associated congenital anomalies. The other organs were not remarkable.

Summary.—Congenital esophageal atresia with tracheo-esophageal fistula. Operation on the fifth day by transection of the upper end of the stomach with the establishment of an abdominal esophagostomy and gastrostomy. Death on the thirty-fourth day, of bronchopneumonia. Autopsy showed small scattered lung abscesses occurring about particles of aspirated barium.

Case 2.—S. B., a male infant, admitted to Strong Memorial Hospital on September 4, 1934, two days after birth. Child was full term and the delivery was spontaneous. There were eight other children in the family, all living and well. When nursing was begun, it was observed that the child took the nipple well and seemed to swallow properly, but almost immediately choked, became extremely cyanotic and ceased breathing. Under fluoroscopic study a nasal catheter was seen to stop completely in the upper esophagus and curl upwards. Skiodan was injected through the catheter and a blind dilated upper esophageal pouch was outlined. The x-ray examination showed a large amount of gas in the stomach and bowel. A diagnosis of congenital esophageal atresia with tracheo-esophageal fistula was considered established. The child was supported with glucose and saline injections and on the following day, September 5th, a laparotomy was performed by Dr. W. J. M. Scott under ether anesthesia. The liver was quite large. The stomach was lifted anl clamped across as close to the esophagus as was possible. It was then cut across and the edges turned in with a double row of sutures. A gastrostomy was then performed. The child withstood the operative procedures satisfactorily. He was supported by infusions and by several small transfusions. The child gained weight slowly and did well until the last of November, when he developed a troublesome diarrhea, which cleared after the addition of lactic acid to the formula. Around the middle of January, 1935, this recurred and the child lost ground rapidly in spite of transfusions and infusions. Death occurred on January 24, 1935, 145 days after birth.

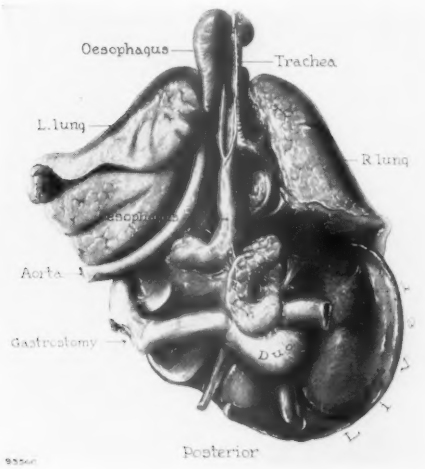

Fig. 3.

Autopsy Findings.—The findings in the study of the esophagus (Fig. 3) were entirely similar to those reported in the previous case. Considerable hypertrophy of the walls of the upper segment was noted. The lower segment opened into the trachea 1 cm. above the bifurcation and was very narrow, 4 mm. in diameter, for a distance of 3½ cm. below this point. The two segments were separated by a distance of 1.5 cm. The lungs showed a purulent bronchitis with a slight interstitial pneumonia and small areas of atelectasis. There were no associated congenital anomalies.

Summary.—Congenital atresia of the esophagus with tracheo-esophageal fistula. Operation on the second day consisting of transection of upper end of stomach combined with gastrostomy. Death on the 145th day.

DISCUSSION

In the surgical management of these cases, the first and fundamental aim must be to prevent regurgitation through the lower esophageal segment into the lungs. This requires closure of the lower segment by ligation or actual transection. On purely theoretical grounds, the most logical point of attack would be the upper portion of the segment near the esophagotracheal junction, following the transthoracic procedures of Richter or Iglauer. There are, however, almost insurmountable practical difficulties to such methods. The difference between the vital capacity and the tidal air of the newborn is exceedingly small and leaves insufficient reserve to permit a radical exposure of the intrathoracic esophagus. To close the lower segment, therefore, without resorting to thorocotomy, compels us to approach it below the diaphragm. It is important to point out in this respect that the relatively large size of the liver of the newborn frequently

interferes considerably with this procedure and necessitates inclusion of a portion of the upper end of the stomach with the esophagus. Ligation is of course the simpler procedure. Occlusion by a silk ligature is of only temporary value, because it will insure closure for a relatively short period—possibly a week or ten days. Transection can be done with or without the establishment of an abdominal esophagostomy. The disadvantage of the latter procedure is that it requires inclusion of a somewhat larger segment of stomach in order to bring the segment up to the abdominal wall without tension. There is also considerable resulting wound infection, as was well illustrated in Case 1. Both ligation and transection alone are objectionable, because secretions from the included segment of gastric mucosa continue to drain into the lung and the danger of pneumonia is not eliminated. Closure of the communication with the trachea is therefore still of prime importance.

The successful rapid production of bronchial stenosis, reported by Cutler and Wood,[21] by the use of 25 per cent solution of aqueous acid acriflavine, made us feel that their technic might be used to advantage in solving this problem. Starting with the knowledge that painless strictures of the urethra were not uncommon following irrigation with acriflavine solution, and that the drug possesses great powers of penetration without being systemically toxic except in large doses, they found it possible to produce complete occlusion of the main bronchus of a dog in about twenty-eight days. Undesired reactions on adacent tissues in the form of hemorrhage, gangrene or abscess formation, so frequently noted in stronger types of sclerosing solutions, were absent. Tucker's demonstration that the fistulous opening of the lower segment into the trachea or main bronchus can be identified and catheterized through the bronchoscope made us feel that the same sclerosing technic of Cutler might be successfully used to close off this communication. It may be recalled at this point that the average diameter of the upper portion of the lower segment is about 3 mm. for a distance of about 4 to 7 cm. before it assumes its normal caliber near the stomach. This fact should make rapid closure by the sclerosing agent relatively easy. With this in mind, it is evident that in preventing fatal regurgitation by the immediate closure of the gastric end of the lower segment while stenosis of its tracheal end is being effected, we must aim to avoid the formation of a blind, undrained secretory segment between the point of closure below and the area of stenosis above. The establishment of an abdominal esophagostomy would of course avoid this but would present the objections already mentioned as well as difficulties for future management. Occlusion of the gastric end of the lower segment with a fascial

strip would seem to our mind to offer important advantages. In the first place it would produce relative occlusion, for several weeks at least, and thus overcome the rapid re-establishment of the lumen noted after ligation with silk. Occlusion in this way might possess the added important advantage of gradually loosening and thus permitting sufficient drainage of the lower segment into the stomach after stenosis of the tracheal end had been accomplished. A permanent gastrostomy opening must of course be made at the time of ligation.

The importance of the upper segment must not be overlooked, as has been emphasized by Lilienthal[22] and more recently by Iglauer. The overflow of accumulated mouth secretions from this blind pouch into the trachea presents a constantly dangerous source of pulmonary infection. Iglauer proposes that cervical esophagostomy be the first step in the management of these cases. "Unless the upper segment can be taken care of, there are practically no indications for any further intervention." Our observations in the two cases reported convince us that the closure of the lower segment is of primary importance and that overflow from the upper segment does not regularly cause the fatal aspiration pneumonia within the first few days of life. We fully recognize the necessity for early external drainage of the upper segment, but feel that by frequent aspiration drainage through the nose and throat this procedure may be temporarily postponed until such time as the patient's condition best permits. Cervical esophagostomy under local anesthesia should be attended with little risk, especially if preliminary packing of the wound to seal off the mediastinum is practiced. The fact that the lower portion of the blind upper segment is usually intrathoracic must be emphasized.

SUMMARY

In the management of the common variety of congenital esophageal atresia with communication between the lower segment and the trachea we would advise:

1. Immediate laparotomy with occlusion of the cardiac end of the stomach by means of a fascial strip accompanied by permanent gastrostomy. This insures adequate feeding without otherwise fatal regurgitation into the lungs.

2. Closure of the fistulous communication with the trachea by means of a stenosis produced by a sclerosing solution, such as aqueous acid acriflavine (sol. 25 per cent), introduced by a swab through the bronchoscope.

3. Temporary management of the blind upper segment by frequent aspiration rather than by immediate surgery. It is to be emphasized, however, that early external drainage is desirable.

We feel that these steps are all surgically possible and eliminate the practically insuperable obstacles to transthoracic methods in the newborn.

11 North Goodman St.

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XCIV

BRONCHIAL OBSTRUCTION IN CHRONIC TUBERCULOSIS*

KENNETH A. PHELPS, M.D.

MINNEAPOLIS

The recognition of bronchial obstruction in patients with pulmonary parenchymal tuberculosis frequently explains clinical phenomena and pathologic pictures which would otherwise remain obscure. Bronchial obstruction may be due to numerous causes: Tuberculosis, aneurysm, cancer, foreign body, etc., any of which may occur in a tuberculous individual. This paper will deal only with bronchial obstruction of tuberculous etiology.

The frequency of this condition is not yet fully appreciated, but its importance has long been realized. The literature of the past few years contains several references to bronchial stenosis in tuberculosis, the article by Eloesser[1] being of particular interest. Throughout this paper I shall repeatedly refer to his work.

There are three types of bronchial obstruction due to tuberculosis.

1. Intramural; the lesion is located within the lumen of the bronchus.

2. Mural; the lesion is located in the wall of the bronchus.

3. Extramural; the lesion is located outside the wall of the bronchus. The site involved may be any point or points in the bronchial tree, from the larynx to the finest bronchiole.

1. *Intramural obstruction* is most frequent in children. It is caused by a suppurating peribronchial gland, usually located near the bifurcation, which breaks through the bronchial wall and sloughs out into the lumen. This necrotic material acts like a foreign body in the bronchus, producing the same symptoms and signs.[2,3,4] It may be coughed out or removed by means of the bronchoscope.

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*Read before the nineteenth annual meeting of the American Bronchoscopic Society, Detroit, May 27, 1936.

REPORT OF CASES

CASE 1.—Ulcerating gland acting like a foreign body. Child, age twelve months. At three weeks of age had a severe cold, followed by a persistent wheeze, intermittent temperature, cough and cyanosis at times. No history of foreign body. Physical examination revealed: findings which suggested the diagnosis of asthma, but adrenalin did not relieve the symptoms. X-ray: after many plates were taken in all phases of respiration, one showed definite obstructive emphysema of the right middle and lower lobes. A diagnosis of nonopaque foreign body was made and bronchoscopy performed. The bronchial orifices were edematous and narrow, and no foreign body was seen. In a few days a second bronchoscopy was done, and granulation tissue from the rupture of a peribronchial gland was seen in the right bronchus. Mantoux test was positive. Child is now in Glen Lake Sanatorium with bone tuberculosis.

In adults the intramural obstruction may be due to cartilaginous sequestra or a broncholith may originate in a peribronchial lymph gland and eventually break into the lumen of a bronchus. At this site granulation tissue may persist or scar tissue form. These lesions are apt to be in the lower bronchi, rather than near the bifurcation, as occurs in children. Eloesser states that mild x-ray therapy is of definite value in enlarged tuberculous glands, though it must be used with extraordinary care, particularly if parenchymal tuberculosis is present.

CASE 2.—H.: Broncholith. Age, 37 years. Cough for five years with considerable sputum, and occasional attacks of hemoptysis since double pneumonia and pleurisy in 1933. The x-ray shows a small amount of fibrosis at the apices, with mottled infiltration on both sides. A calcified area is present at the level of the fourth rib anteriorly. Sputum negative for tubercle bacilli. After coughing up an irregular stone, the size of a pea, the patient was markedly improved.

Bronchoscopy about one week later: no remnants of stone were found and the area of rupture could not be located. A small amount of purulent secretion was aspirated.

2. *Mural Stenosis.*—The pathology of this type of obstruction can be divided into three stages:

(a) Submucous tuberculoma with infiltration, thickening and edema of the bronchial wall.

(b) Superficial ulceration of the mucosa, eventually replaced by granulation tissue.

(c) Cicatricial stenosis of the bronchial lumen having a similar appearance to a lye stricture of the esophagus.

CASE 3.—M. N., age 25. In 1931 complained of cough and weakness. X-ray of the chest showed tuberculosis. Patient took no treatment and continued at work until May 17, 1934, when hemoptysis, about one glass full, occurred. Admitted to Glen Lake Sanatorium May 30, 1934. X-ray at this time showed probable ob-

structive emphysema of the left lung. Pneumothorax January 12, 1935, to date. Loud wheezy respiration was noted. Bronchoscopy September 8, 1935. Mucosa of the left main bronchus is red and granular with ulceration and granulation tissue in the stem bronchus. Lipiodol demonstrates the stenosis of the left bronchus. Bronchoscopy repeated April, 1936, and a cicatricial stricture of the left main bronchus without ulceration or granulation tissue was found. Very little pus could be aspirated from the distal bronchi. Following aspiration the stricture closed, atelectasis developed, the wheeze disappeared and for a few days the temperature was elevated. Gradually the edema subsided and patient returned to her previous condition. This patient has been observed for five years by x-ray and bronchoscopy, from the early stage of tuberculosis, followed by ulceration of her bronchial mucosa, with the development of granulation tissue and ending in an annular stricture of her bronchus.

3. *Extramural.*—Compression stenosis is most frequently due to the pressure of enlarged peribronchial tuberculous glands. It may occur in children or in adults. Rarely interlobar empyema or paravertebral abscess may rupture into the mediastinum and cause bronchial compression. Peribronchial sclerosis or parenchymal fibrosis will at times kink or distort the bronchi and thus create an obstruction.

The pathologic changes produced by obstruction of the air passages are well known to the members of this society. However, many of our medical friends are not so familiar with them. We should discuss obstructive emphysema, obstructive atelectasis, drowned lung, etc., at every medical opportunity. On the other hand, I suspect that bronchoscopists may be less well posted on the secondary effects of stenosis, such as its relation to the heart and larger blood vessels.

The symptoms of obstruction depend upon its site and degree, as well as its duration. Tracheal narrowing is characterized by an audible stridor, extreme dyspnea, with suffocation appearing after slight exertion. Bronchial stenosis may cause a pain or a pulling sensation in that side of the chest. Lower sites are quite apt to cause retention of secretion, with resulting toxemia. Partial obstruction produces wheezing and rattly breathing, with a dry irritating cough. Perhaps the asthmatic symptoms, which are occasionally associated with tuberculosis, may be actually due to a stenotic bronchus. The symptoms of complete obstruction depend upon the condition of the distal portion of the lung. If no suppuration is present, the symptoms are quite insignificant, but if suppuration is present or develops, the symptoms of toxemia occur promptly. It is well known that sudden stenosis causes more violent symptoms than a slowly developing one.

The diagnosis of stenosis can frequently be made by means of the x-ray, particularly after lipiodol is instilled. The x-ray will show

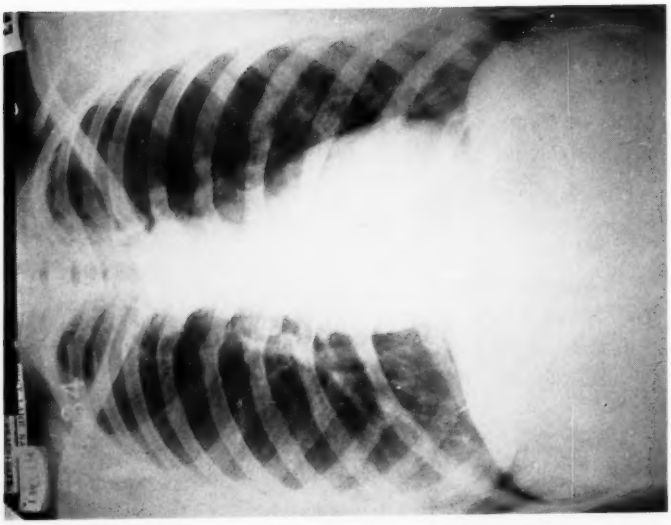

Fig. 2. Case 3, 1934. Emphysema left lobe, first sign of bronchial obstruction. Probably ulceration.

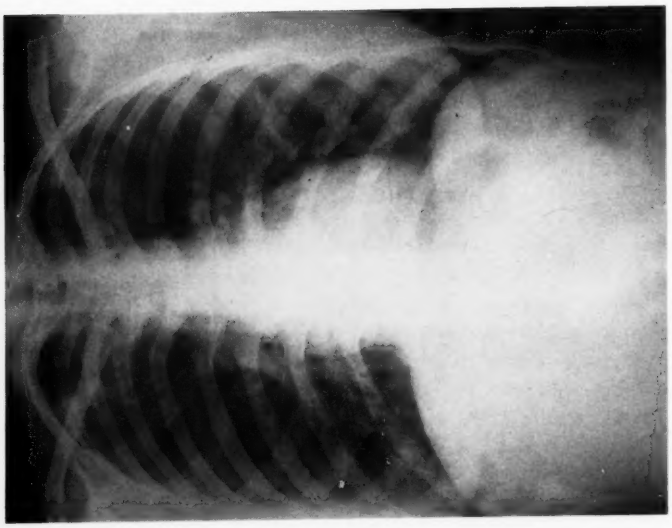

Fig. 1. Case 3, 1931. Early pulmonary tuberculosis. No bronchial obstruction.

obstructive emphysema or atelectasis, but will not always differentiate their etiology. In tuberculous lungs, which have been long compressed by pneumothorax, atelectasis may be followed by fibrosis. It is very difficult, by clinical methods alone, to make the differential diagnosis between diffuse fibrosis, in such cases, and atelectasis due to bronchial obstruction. Both conditions may have similar physical findings: dullness, flatness, limitation of expansion and contraction of the chest wall. The x-ray shows both as a uniform denseness associated with deviation of the trachea, mediastinum and the diaphragm. Even with lipiodol the x-ray evidence may be inconclusive. Bronchoscopy is the most satisfactory method of making the diagnosis (3) as nothing can compare with direct inspection of the bronchial orifices. In tuberculosis, the most important lobe is the upper lobe, and this is the one most difficult to examine bronchoscopically.

When a straight, rigid tube is passed through a collapsed or deformed bronchus, one may get the impression that there is no obstruction present or it may appear that the obstruction is at one level, while it actually is at another. Intermittent stenosis is most probably due to plugs of dry secretion, and in our experience is not commonly seen in tuberculosis.

At the last meeting of the American Association for Thoracic Surgery, Sampson of Ann Arbor reported a series of twenty-four cases of tuberculous tracheobronchitis. Seventeen were of the stenotic type, and at the end of one year 50 per cent had died and the others showed no improvement. He stated that results seem equally poor, whether or not active collapse therapy is instituted. We do not feel that the outlook is quite so hopeless.

The rare condition of complete occlusion of a single lobe bronchus, uncomplicated by distal infections, needs no treatment. The loss of the function of one lobe is not of great importance, and it is possible that the stenosis may cure the tuberculosis in that lobe by permanently putting it out of commission.

The problem, by no means completely solved, is what to do for partial stenosis. The use of electrocoagulation in the ulcerative stage seems hopeful. Dilating strictures with bougies is at times of considerable benefit; however, the process is tedious. According to Eloesser, collapse therapy may convert a partial obstruction into a total occlusion so that a poorly draining suppuration may be completely blocked off. Symptoms of toxicity will appear and bronchiectasis or abscess will be produced. We have seen no case in which we felt that collapse therapy was the cause of bronchiectasis. We believe that in such

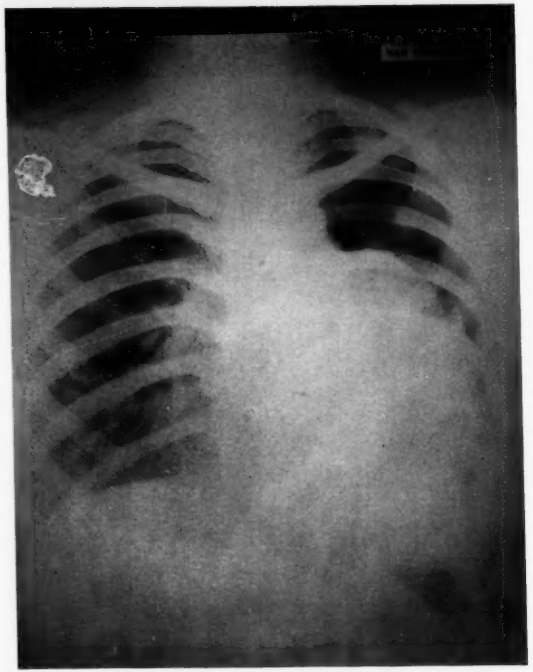

Fig. 3. Case 3, 1936. Two days following last bronchoscopy. The slight trauma of inserting an aspirating tip through the stricture, produced enough local reaction to occlude the bronchus and cause atelectasis.

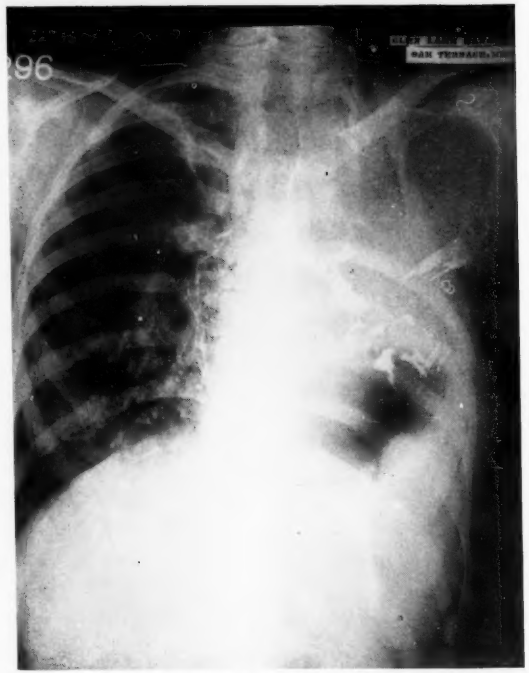

Fig. 4. Case 10. Bronchial stricture with bronchiectasis. Results of thoracoplasty not satisfactory. Bronchoscopy gave patient considerable relief.

cases bronchiectasis was probably present, even though unrecognized, before such treatment was instituted, and accounts for the unsatisfactory results obtained. If bronchial ulceration is active, it seems to us that collapse therapy may be of benefit, just as it helps tuberculous laryngeal ulceration.

In our experience the most frequently found obstruction is granulation tissue in the stem bronchi, even though the parenchymal lesion is nearly always in the upper lobe. Suppuration, then, occurs distal to the stenosis and bronchiectasis develops, which may be tuberculous or not. Thus a patient may have upper lobe tuberculosis with associated lower lobe bronchiectasis of a nontuberculous nature. Further pathologic observation will be necessary to substantiate this idea, as biopsies taken from our patients have not yet revealed positive information.

The following cases will demonstrate the above mentioned sequence of events:

CASE 4.—Mrs. H., age 32. Admitted 1933. Sputum positive. X-ray shows A large lymph gland causing extramural obstruction with atelectasis of the right upper lobe. Pneumothorax instituted and continued. Intrapleural pneumonolysis, 1934. In spite of good pneumothorax, with no uncollapsed cavity, the patient persisted in having severe attacks of coughing, with increase of sputum from very scant to about two ounces. These attacks recurred every four weeks, and she felt fine in the intervals. Bronchoscopy showed the right main bronchus obstructed by granulation tissue. Lipiodol was injected into this bronchus and definite bronchiectasis was demonstrated. Patient improved and bronchoscopy was repeated with further improvement. Sputum is now negative.

This case started with atelectasis of the right upper lobe, which increased in spite of treatment. Bronchoscopy demonstrated the presence of granulations in the right main bronchus, producing bronchiectasis in the lower lobes.

CASE 5.—H. N., age 24, admitted 1933. Sputum positive. X-ray: Fibroid left and right negative. Pneumothorax since 1934. Paroxysmal cough with wheezing and noisy respiration. Bronchoscopy: Redness and thickness of the mucosa at left main orifices, which bleeds easily, with granulations extending into the lower lobe bronchus. Ten per cent silver nitrate applied and considerable pus aspirated. The wheezing has disappeared and the cough was relieved. Cough recurred and again relieved by bronchoscopy. Last treatment September, 1935, and relief has persisted. This patient has not yet developed bronchiectasis and perhaps bronchoscopy will help prevent its development.

CASE 6.—M., age 27, admitted 1934. Sputum positive. X-ray: left negative, cavitation right upper lobe. Pneumothorax on admission. Phrenephraxis March 19, 1935. Right intrapleural pneumonolysis May 21, 1935. Cough and expectoration continued in spite of good pneumothorax. Bronchoscopy September 26, 1935: Considerable granulation tissue in the right main bronchus. This was removed by forceps and silver nitrate applied. Cough and expectoration improved. Broncho-

scopy repeated with improvement after each treatment. This case has no bronchiectasis as yet.

CASE 7.—S. P., age 28, admitted May 26, 1934. Chronic cough, malaise and hemoptysis since 1927. X-ray on admission: Right lung clear, left fibrotic upper lobe with cavitation. Bronchiectasis lower lobe. Bronchoscopic examination, June 1, 1934: Orifice of the left lower lobe bronchus appears to be normal. Patient left hospital to move to different climate and no follow up was possible. This case is one of upper lobe tuberculosis and lower lobe bronchiectasis without any visible bronchial obstruction. Possibly this is an example of tuberculous bronchiectasis of the lower lobe.

Bronchoscopy is of therapeutic value when the patient has recurring attacks of retention producing fever and other signs of toxicity, even though some form of thoracoplasty has been performed. Bronchiectasis of the bronchi in the collapsed lung will be found in some of these cases, and great help may be obtained by bronchoscopic aspiration.

CASE 8.—B., age 18, on admsision in 1925. Thoracoplasty in 1930. About once in three months she would have attacks of coughing and expectoration, with temperature as high as 104, lasting for three weeks. Bronchoscopy January 18, 1934: Considerable thick yellow pus was removed from the right bronchi. Lipiodol shows marked bronchiectasis. Since bronchoscopy she has had no attacks of coughing or temperature and is doing full time work. Bronchoscopy has been repeated every three months, six in all, with no recurrence of her previous attacks.

CASE 9.—F. M., age 19 on admisison July 14, 1930. Sputum positive. X-ray: Multiple cavitation right apex and bronchiectasis right base. Thoracoplasty first stage March 16, 1933; second stage May 25, 1933. The lower part of the lung was not compressed because of the recognized bronchiectasis. Following two-stage thoracoplasty she continued to have excessive cough and expectoration up to four ounces. Bronchoscopy November 13, 1934: Right bronchus obstructed with granulation tissue. Pus aspirated and lipiodol injected. Following bronchoscopy cough and expectoration decreased and patient definitely improved. Bronchoscopy repeated every few months with improvement.

CASE 10.—E. R., age 24, admitted January 25, 1928. Sputum positive. X-ray: Advanced bilateral tuberculosis with cavitation left upper. Pneumothorax January 20, 1931, to August 22, 1931. Left phrenephraxis March 11, 1930. Three-stage thoracoplasty 1934 to 1935. Recurrent attacks of sputum retention, with excessive cough and expectoration with temperature to 103. Bronchoscopy December 5, 1935: Granulation tissue throughout left stem bronchus. Large amount of pus aspirated and lipoidol shows bronchial stenosis and bronchiectasis. Some improvement in drainage following bronchoscopy, but symptoms recurred. Further treatment will be continued.

CASE 11.—H. B., age 23, admitted April 23, 1931. Sputum positive. X-ray: Extensive pulmonary tuberculosis of left lung with cavitation in upper lobe. Pneumothorax May 15, 1931, to April, 1932. Five-stage thoracoplasty 1932 to 1933. Bronchiectasis diagnosed at operation. Left phrenephraxis June 1, 1933. Left

phrenicectomy November 21, 1933. Continued cough and expectoration, with bloody streaking up to two ounces. After complete surgery, she continued to have intermittent attacks of retention, with temperature to 104. Bronchoscopy showed stenosis of the left main bronchus. Lipiodol revealed bronchiectasis.

It seems reasonable to suggest that a patient, suspected of having stenosis, should be bronchoscoped before collapse therapy is instituted. Compression of a lung that has a partially blocked bronchus with threatened bronchiectasis, may hasten the development of sepsis and therefore such treatment will probably be ineffective. On the other hand, a direct surgical attack on intrapulmonary tuberculous suppuration is not casually undertaken, but may be indicated when the bronchoscope demonstrates a stenosis with persistent purulent secretion, and repeated bronchoscopic treatment is unsuccessful. The following case illustrates this point.

CASE 12.—M. O., age 34, on admission August 10, 1933. Sputum positive. X-ray: Entire right lung involved with extensive cavitation. Pneumothorax September 26, 1933, to January 1, 1935. Following this, attacks of coughing and temperature up to 105, loss of weight, malaise, secondary anemia and poor general condition. Bronchoscopy December 13, 1934: Right main bronchus obstructed with granulation tissue. Large amount of bloody purulent secretion aspirated. Marked improvement followed, with decrease in temperature and cough, sputum easier to raise, gain in weight and general prognosis better. Bronchoscopy repeated ten times during the next year. Very little pus was obtained the last few times, and granulations still obstruct the bronchus. The patient's symptoms were not improving, so an external operation was decided upon. A cautery lobectomy was done. Since this operation her sputum has been reduced to only a few cc. daily, her temperature is normal, her hemoglobin has gone up 25 per cent, her amenorrhea has disappeared.

CONCLUSIONS

1. Bronchoscopy has helped teach us that tuberculous bronchial stenosis is quite frequent in chronic pulmonary phthisis.

2. In children a peribronchial gland may rupture into a bronchus and then bronchoscopy may be a life saving procedure.

3. In adults bronchoscopy enables one (a) to remove a broncholith, (b) to diagnose ulceration and treat it, (c) to diagnose scar formation and treat it.

4. The bronchoscopist can aid the thoracic surgeon decide upon the type of operation he will perform, depending upon the presence or absence of bronchial stenosis.

5. When operation does not give the patient complete relief, the bronchoscopist can frequently make him more comfortable.

6. Lower lobe nonspecific bronchiectasis is a frequent complication of pulmonary tuberculosis in the upper lobe, and develops as a result of tuberculous bronchial obstruction.

7. Since information revealed by bronchoscopy is of such importance and the results of bronchoscopic treatment are so encouraging, one can safely say, "No good sanatorium for tuberculosis is completely staffed without a bronchoscopist."

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XCV

THE BIPLANE FLUOROSCOPE AS AN AID IN BRONCHOSCOPY*

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AND
W. EDWARD CHAMBERLAIN, M.D.

PHILADELPHIA

HISTORY

As early as 1907, pioneer work in fluoroscopically guided bronchoscopy was begun by Grier in Pittsburgh, working with Chevalier Jackson and Ellen J. Patterson. Jackson[1] discussed the subject fully in his textbook, "Peroral Endoscopy," published in 1914, calling attention to the fact that "fluoroscopic bronchoscopy is so deceptively easy from a superficial theoretical point of view that it has been used unsuccessfully" in cases that would have been "easily handled in the regular endoscopic way." He stated that "in a collected series of twelve cases the object was removed in 66.7 per cent with a mortality of 41.6 per cent, and concluded that, "because of its high mortality and low percentage of success," fluoroscopically guided bronchoscopy "was not justified in any case of bronchially lodged foreign body until after regular ocularly guided bronchoscopy had failed." It must be remembered that the best fluoroscopic apparatus available in 1914 was very inferior to our present day equipment, and visibility on the fluoroscopic screen as well as visualization in roentgenograms was much less efficient. In many cases a foreign body was not visible on the fluoroscopic screen, but in these cases some aid in localization was obtained by taking roentgenograms with an endoscopically introduced opaque probe in various branch bronchi. In 1916, Grier[2] published an excellent paper on "Fluoroscopic Bronchoscopy," in which he presented an illustrated description of the apparatus he was using at that time. Manges[3] worked for years with Chevalier Jackson in perfecting the technic of bronchoscopy with biplane fluoroscopic guidance, and his paper, published in 1933, presented his views on the subject. The year before Manges' article appeared, an excellent paper was published by Pancoast and Pendergrass,[4] with a note by

*Read by Chevalier L. Jackson at the nineteenth annual meeting of the American Bronchoscopic Society, Detroit, Michigan, May 27, 1936.

Fig. 1. Film taken on the biplane fluoroscope, showing a common pin grasped by its mid-portion. With a grasp such as this it is sometimes best to bend the pin into the end of the costophrenic bronchoscope. In this particular case, however, it was found possible subsequently to grasp the point and remove the foreign body through the tube. (See Fig. 2.) Film to the left shows the anteroposterior view and film to the right the lateral. Both films in this and subsequent figures are placed in a position to simulate the appearance on the screen from the bronchoscopist's point of view.

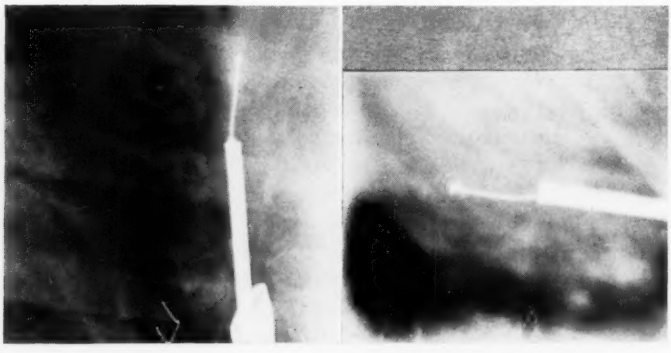

Fig. 2. Films taken on the biplane fluoroscopic table, showing a common pin about to be drawn into the costophrenic bronchoscope (Same case as Fig. 1).

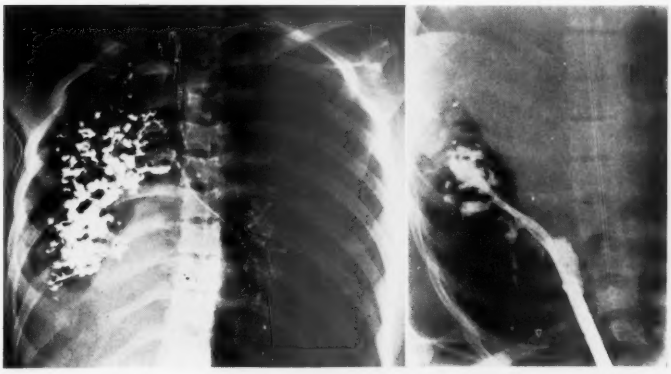

Fig. 3. Bronchogram made by fluoroscopically guided bronchoscopic bronchography. Because of the high degree of obstruction produced by a tumor of the right main bronchus it was impossible to fill the right upper lobe with lipiodol except by introducing a flexible tipped metal tube through a bronchoscope. Film to the right was taken on the fluoroscopic table and is placed upside down to simulate the view obtained by the bronchoscopist.

Tucker. Jackson's later textbook, "Bronchoscopy and Esophagoscopy"[5] (1922, 1927 and 1934), gives a rather full discussion of fluoroscopically guided bronchoscopy and includes some important points of technic. While still warning of the dangers of single-plane fluoroscopic guidance, the author points out the need for *double-plane* fluoroscopic guidance, and states that "a tribute is due Dr. Willis F. Manges for his marvellous development of fluoroscopic apparatus and fluoroscopic co-operation, which have practically eliminated the impossible in bronchoscopy for slender foreign bodies, such as pins, in minute bronchi at the periphery of the lung." The invaluable aid of the biplane fluoroscope in "costophrenic bronchoscopy" is convincingly demonstrated in an article by Jackson and Jackson[6] entitled "Pins at the Periphery of the Lung," and the whole subject of fluoroscopically guided bronchoscopy is discussed in detail in two recent textbooks[7,8] by the same authors. Equen[9] reported his experiences with the biplane fluoroscope in a paper read before the American Laryngological, Rhinological and Otological Society about two years ago.

INDICATIONS

1. In considering the indications for the use of biplane fluoroscopic guidance in bronchoscopy today, we think first, of course, of foreign bodies in the periphery of the lung, especially common pins

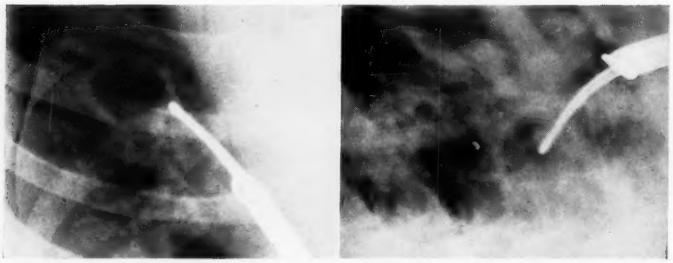

Fig. 4. Films taken on the biplane fluoroscopic table to record the relations of a certain branch bronchus to a cavity in a tuberculous patient with artificial pneumothorax in whom bronchoscopic bronchostenosis was contemplated. Note that the anteroposterior view (on the left) shows the tip of the opaque bougie not quite entering the cavity, though the lateral view (on the right) suggests that it has entered.

(Figs. 1 and 2), to be removed by costophrenic bronchoscopy. Any opaque foreign body lodged in the peripheral portion of the lung constitutes an indication for biplane fluoroscopic bronchoscopy. In a recent paper[10] on foreign bodies one of us (C. L. J.) reported, with illustrations, a case in which a fragment of tooth was lodged in a small peripheral branch bronchus. The removal of this foreign body was made possible only by expert biplane fluoroscopic assistance. It must be borne in mind that while the guidance of the biplane fluoroscope may make an otherwise impossible case possible, it does not always make it easy, and very often patient, careful work for an hour or more, or even a second bronchoscopy may be required before success is attained. Biplane fluoroscopic guidance is of value in the removal of screws and nails lodged head upward, since fluoroscopic guidance enables us to determine whether or not our forceps blades open widely enough, and whether or not we are allowing them to open too widely. Any large or sharp pointed object is handled more satisfactorily if the guidance of the biplane fluoroscope is used. Foreign bodies in the stomach, whether removed gastroscopically or with the Tucker flexible forceps, can be removed only with fluoroscopic guidance.

2. The introduction of lipiodol for bronchography in cases of bronchial stenosis is best accomplished by fluoroscopically guided bronchoscopic bronchography (Fig. 3), though for routine bronchography we prefer one of the indirect catheter methods, using the fluoroscope as an aid in the accurate placement of the catheter.

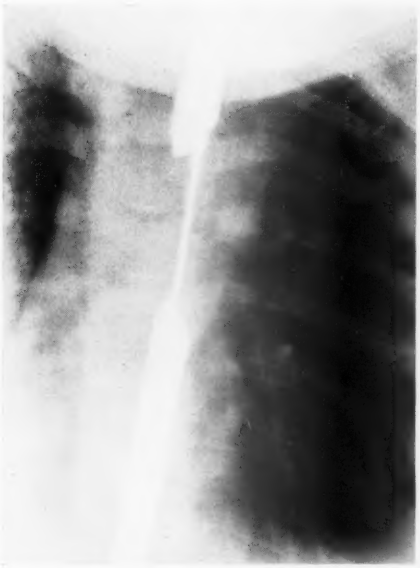

Fig. 5. Film taken on the bilplane fluoroscopic tube, to show the relative positions of the peroral and retrograde esophagoscopes and the opaque bougie used to "thread the esophagus" in cases of tight stricture or complete atresia, preliminary to dilatation by peroral or retrograde bouginage.

3. Though "costophrenic bronchoscopy" is generally thought of chiefly as a method of removing foreign bodies from the periphery of the lung, the exploration of peripheral bronchi in order to find the one leading to an area of density in the outer lung field with a view to biopsy or the tapping of an abscess cavity is also "costophrenic bronchoscopy," and may often be done to advantage under the guidance of the biplane fluoroscope (Fig. 4).

4. Another important use the endoscopist makes of the biplane fluoroscope is to guide him in the procedure of combined peroral and gastrostomic esophagoscopy for "threading of the esophagus" (Fig. 5) in cases of tight stricture or total atresia. This procedure is always a hazardous one, but if the greatest care is exercised, fluoroscopic guidance will increase the number of these cases in which a thread may be successfully introduced for use as an aid in subsequent treatment of either peroral or retrograde bouginage.

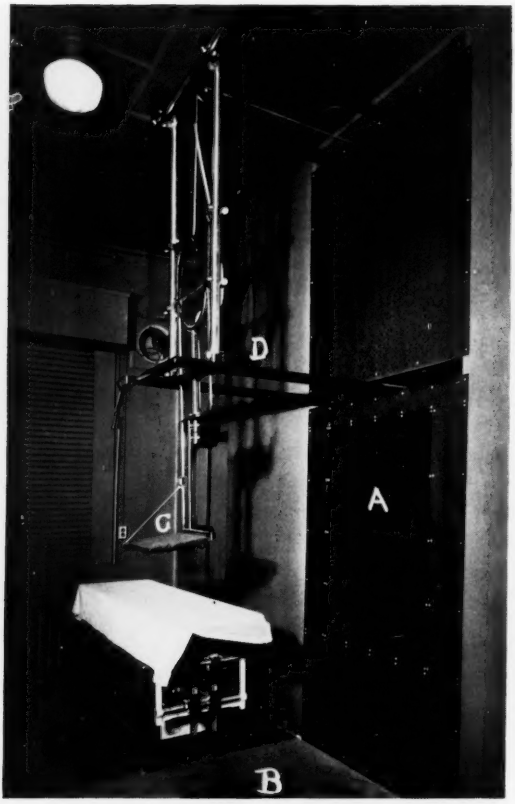

Fig. 6. The Chamberlain biplane fluoroscope. The tube for the horizontal ray is back of the aluminum panel, *A*. The tube for the vertical ray is beneath the floor, under the bakelite panel, *B*. Both tubes and the fluorescent screen, *C*, are carried on an integral supporting frame. By this arrangement both the horizontal ray and the vertical ray are always centered on the fluorescent screen, wherever the screen may be placed during the course of an examination.

THE CHAMBERLAIN BIPLANE FLUOROSCOPE

When the roentgenologic co-author, Edward Chamberlain, first became associated with Temple University, about five years ago, the matter of biplane fluoroscopic guidance in bronchoscopy was discussed with him. He was told what had been the chief shortcomings of the various types of apparatus used previously, and he set to work

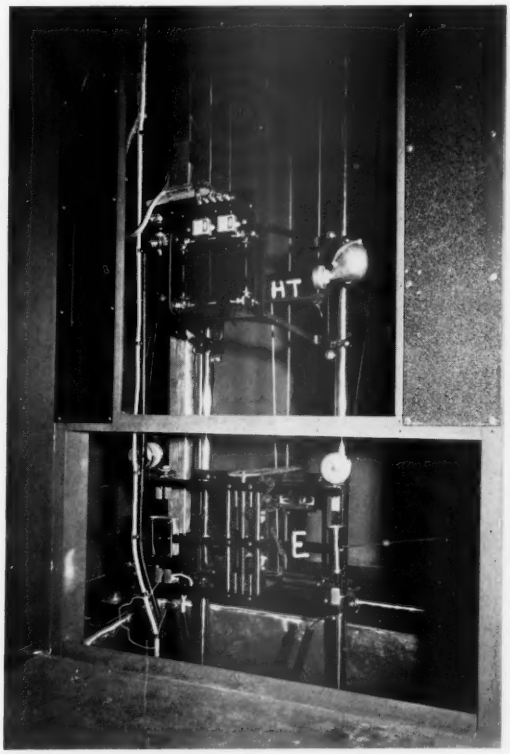

Fig. 7. Close-up view of horizontal ray tube, *HT*, after removal of aluminum plate (*A*, Fig. 6). In the lower portion of the picture is shown *E*, the motor-driven high-tension switch which permits remote control of the change-over from vertical ray to horizontal ray fluoroscopy.

to design a new fluoroscope which would overcome them. For example, it had always been difficult for the head-holder to support the patient's head in the proper position and yet not be crowded by the horizontal ray tube or its supports. Therefore, Chamberlain substituted a tube at a distance of four feet, behind an aluminum panel (Figs. 6 and 7), with the added good effect that distortion was reduced with no loss of visibility, but rather increased perception of detail. As an additional improvement, both tubes were mounted on a frame of steel tubing which traveled on rails, and on this framework also the single screen used alternately in the two planes was mounted, with the result that it moved at all times exactly with the tubes.

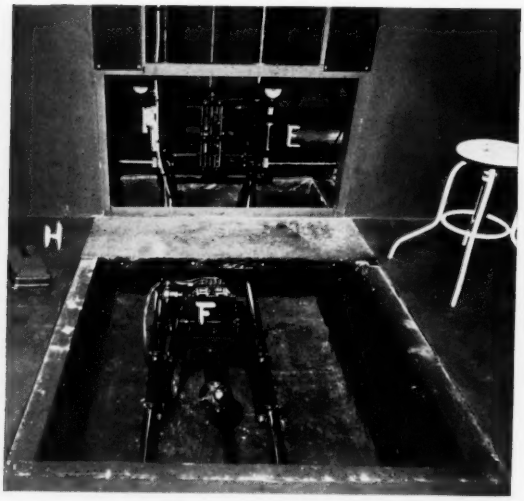

Fig. 8. View of vertical ray tube *F*, after removal of steel floor-plate and bakelite panel (*B*, Fig. 6). Note that this tube is mounted on the same integral frame which carries the horizontal ray tube and the support of the fluorescent screen. At *E* is shown the remote controlled high-tension switch (Cf. Fig. 7).

At first the original old tube under the table was used for the vertical ray, but in our present apparatus the tube for the vertical ray is sunk under the floor, also at a distance of four feet from the patient (Fig. 8). A "transom latch" mechanism is used to hold the screen in the horizontal position when used with the vertical ray. Separate foot switches mounted upon a common base control the horizontal and vertical ray units.

An important feature of the Chamberlain apparatus is the pair of automatic self-starting electric clocks (Fig. 9) which record the exact duration of exposure of the patient's skin to the vertical and horizontal rays respectively. These two x-ray beams having been previously calibrated by means of the ionization chamber, the total permissible exposure time for either beam is known in advance, and the possibility of overexposure or unnecessary cutting short of the procedure are thus both eliminated.

Roentgenograms may be taken with either the horizontal or the vertical ray tube. (Figs. 1 to 5.) It may be of value in studying a difficult mechanical problem to record the relation of instruments to a foreign body (Figs. 1 and 2). Occasionally an indication arises

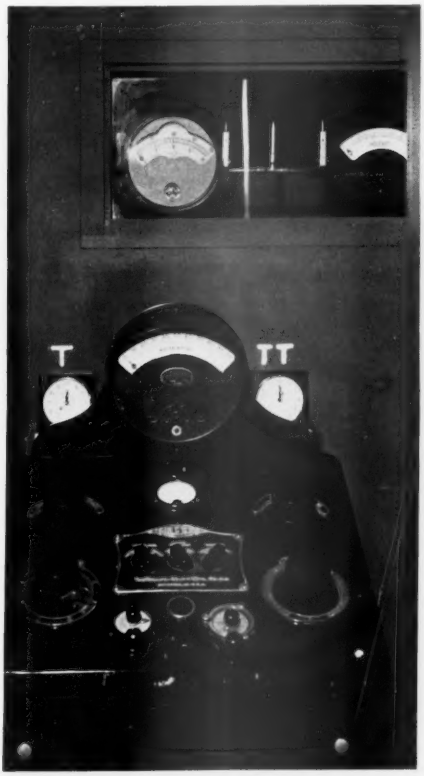

Fig. 9. At *T* and *T T* are shown the self-starting "telechron" clocks for timing the exposure to horizontal and vertical beams respectively. At the beginning of each fluoroscopic procedure the hands are turned to 12:00 o'clock, and no matter how many times the exposure is interrupted or changed from one beam to the other, these locks will indicate the total exposure time for each beam. Accurate ionization chamber calibrations of both x-ray beams enable the radiologist to determine accurately, in advance, the maximum safe time limit for each beam.

for the taking of films immediately after the introduction of a small quantity of lipiodol into a particular portion of the bronchial tree (Fig. 3). In other cases it may be desired to record the relation of instruments to a cavity (Fig. 4) or some other lesion.

CONCLUSIONS

We have thought it worth while to present a discussion of biplane fluoroscopic guidance as an aid in bronchoscopy, because of

the widening indications for it, and particularly because we feel that the fluoroscopic apparatus herein described possesses certain distinct advantages for bronchoscopic work:

1. Complete protection of patient and attendants against the possibility of electric shock.

2. Absence of any interference with the work of the bronchoscopist or his assistants.

3. Maximum protection of patient, radiologist and others present against exposure to x-rays.

4. Central rays of both beams geometrically set at 90° apart, truly vertical and truly horizontal, respectively.

5. Minimum of "enlargement" and "distortion" of the fluoroscopic images. (The distortion inherent in the production of shadow images is materially reduced by increasing the distance between tube and screen while maintaining the screen as close as possible to the patient.)

6. Ease of manipulation and centering, and instantaneous change over from vertical ray to horizontal and vice versa.

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XCVI

MYCOTIC INFECTIONS FOUND IN THE TRACHEA AND BRONCHI*

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It is very interesting to find that the science of mycology, a subject devoted to the investigation and study of fungi, originated long before the discovery of bacteriology.

In 1677, Hooke became interested in the peculiar yellowish spots so frequently found upon rose leaves, and with a crudely constructed magnifying glass was able to make out fungi with filamentous markings, which he later described and illustrated.

Following this original observation many scientists became interested in this subject, and quite a number of early papers were written on fungus diseases occurring in plant life.

Mycology, as a cause of parasitic affections in man, first attracted attention in 1839, when Langenbeck discovered the cause of thrush.

Very soon after this, Schonlein announced the fungus of favus. These reports resulted in other investigations as to the possibility of mycotic causes of many obscure diseases.

In 1844, Gruby found that ringworm was produced by a parasitic fungus, and two years later Eichstedt discovered the etiology of pityriasis versicolor.

Following the brilliant discoveries of bacteriology by Pasteur and Koch, interest in mycology faded into the background for a while, but in recent years much has been written on this subject.

Bronchomycosis, affecting the trachea, bronchi and lungs, may be due to a variety of species of the mycotic family. Some of the most common fungus diseases of interest to bronchoscopists are aspergillosis, torulosis, moniliasis, sporotrichosis, blastomycosis, actinomycosis and coccidioidosis.

Mycotic diseases are most frequently found in tropical countries. Castellani[1] has contributed more to the subject than any other Ameri-

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can investigator. He has traced the occurrence of this infection in man from India to the southern part of the United States and has discovered many pathogenic parasites heretofore unknown. But it should be recognized that these diseases are to be found at times in the colder climates. It is interesting to note that the original etiology of many fungus infections was first discovered in Baltimore.

In 1898, Scheneck[2] was the first to isolate the fungus of sporotrichosis. Boggs and Pincoffs[6] reported the first case of pulmonary moniliasis to occur in this country in 1915.

In 1846, Gilchrist[4] first described and confirmed the cause of blastomycosis. While one of the first cases of coccidioides to occur in North America was reported by Rixford and Gilchrist[7] in the Johns Hopkins Hospital in 1894. Many cases of mycotic diseases have been found in Baltimore, and we now know that such infrequent conditions are to be considered in all undiagnosed pulmonary infections. Most of the infections which involve the pulmonary system produce symptoms closely resembling those found in tuberculosis and from which a differential diagnosis must be made.

The disease has an insidious onset, associated with anorexia, malaise, loss of weight, evening temperature, night sweats and frequent attacks of severe coughing spells accompanied by bloody streaked sputum. The tendency to hemorrhage is a significant sign of this disease. In such cases of hemoptysis when tubercle bacilli cannot be found, fungus infection should always be suspected.

Most of these cases are sent into sanatoria with suspected tuberculosis. At the onset, symptoms, progress and physical findings are so much alike in the two diseases, a diagnosis cannot be made by the usual means. X-ray studies are undependable, and bacteriologic smears and cultures obtained from coughed up sputum may be contaminated by organisms from the mouth, teeth and tonsils. So in the final analysis a positive diagnosis can be made only by bronchoscopic investigation. Exudate must be obtained from the bronchi, so that cultures can be made free of infecting organisms associated with the upper respiratory tract.

Thus adding another group of diseases whereby the art of bronchoscopy is depended upon to make a final diagnosis when all other methods of investigation have failed.

This also demonstrates the importance of having a bronchoscopic clinic connected with tuberculosis sanatoria, a subject I called attention to several years ago. Our work has been not only interesting, but of great practical value, because most of these cases would have remained unrecognized if bronchoscopic studies had not been made.

It is also important, so far as recovery of the patient is concerned, because the successful treatment of mycotic infections depends upon large doses of potassium iodide, a drug definitely contraindicated in tuberculosis.

In this paper no attempt is made to thoroughly cover all phases of mycology, but to briefly consider some of the peculiarities of the most common diseases, as related to bronchoscopy.

ASPERGILLOSIS

Aspergillosis is most frequently found. The niger species predominates, but many other varieties have been reported.

This infection appears to have a definite affinity for involvement of the pulmonary system. The pathology is characterized by the appearance of granulomatous nodules in the mucosa which run a chronic course. The first case was described by Bennet as early as 1842.

Occupation has a distinct relationship to the etiology of aspergillosis. It occurs most often in bird fanciers, pigeon breeders, hair sorters, millers and in people who inhale dust, grain particles and those who are constantly exposed to any form of dry or powdered vegetable matter. The onset of the infection is insidious in character, accompanied by cough, asthenia, anorexia and an evening temperature, and in the early stages is frequently treated as a chronic bronchitis. Later in the chronic course of the disease there is loss of weight, night sweats and hemoptosis, the typical symptoms found in tuberculosis, from which it must be differentiated. Physical findings are uncertain and x-ray examinations show about the same picture in each condition.

There are two symptoms, however, which are of value. A patient suffering with aspergillosis does not look as emaciated and ill as one affected with tuberculosis over a corresponding period of time. The most suggestive sign of all is the frequent tendency to hemoptysis, unassociated with finding tubercle bacilli in the sputum. Finally a definite diagnosis can only be made by aspirating exudate from the affected area through a bronchoscope, uncontaminated by secretions from the mouth and pharynx. In this way pure cultures can be obtained and the true organism discovered by bacteriologic means. The course of the disease is chronic in the nature and the prognosis is fair, provided the case is treated early with iodides.

TORULOSIS

Torulosis has a tendency to invade the cerebrospinal system, primary infections of the lower respiratory tract being extremely

rare; in fact, a case reported by Sheppe[5] is the only one of a primary lesion to be found in the literature. However, secondary complications involving the pulmonary system are very common, occurring in about 42 per cent of cases.

The signs and symptoms of this disease resemble tuberculosis, but headaches, vertigo, dizziness and other indications of cerebrospinal involvement are helpful in making a differential diagnosis. Final determination of the disease depends upon bacteriologic study and finding torula organisms in the affected tissues.

The disease runs a chronic course and the prognosis is fairly good. Localized lesions should be treated by radical surgical procedure. Resection, endothermy and the actual cautery have been used.

The internal administration of colloidal copper has been used with some success in systemic torulosis.

COCCIDIOIDES

Coccidioides, often called California disease, is peculiar because it is so often found in inhabitants of this State. In about 80 per cent of the patients developing this infection it has been found that they had lived in California.

Pulmonary complications are very common because it is believed that the original infection originates in the respiratory tract. Most of these cases give a history of primary symptoms developing in the trachea and bronchi. In advancing stages of the disease, the cerebrospinal system becomes involved with symptoms related to the nervous system.

As in most fungus infections, the pulmonary symptoms resemble those of tuberculosis, but in comparison to the typical symptoms of aspergillosis, where this disease is characterized by early expectoration of profuse amounts of bloody streaked sputum, true hemoptosis is uncommon in the beginning of coccidioides infections.

The course of the disease is progressive in character. In most cases the prognosis is fairly good if recognized in the early stages.

Many remedies have been tried in an effort to alleviate this condition, but no specific cure so far has been found. Injections of colloidal copper apparently give the best results and should be tried in most cases.

SPOROTRICHOSIS

Sporotrichosis involving the pulmonary system has been somewhat unusual, as only six cases had been reported up until 1912.

These cases had been collected by de Beurmann and Gougerot. Since 1912 and 1922 only three more cases have been recorded.

BLASTOMYCOSIS

Following Gilchrist's discovery of blastomycosis, which was reported in 1894, numerous cases have occurred in the literature. While this was originally described as a disease affecting the cutaneous tissues, it is interesting to find that about 95 per cent of cases show complications of the respiratory system. When the infection primarily attacks lung tissues, it runs a rapidly fatal course, but if secondary to a cutaneous lesion the symptoms are mild and of a chronic nature.

ACTINOMYCOSIS

Actinomycosis rarely affects the pulmonary system as a primary lesion. It only invades the trachea and bronchi as an extension from infected tissue above. In the bronchi, fungi and purulent exudate may be the characteristic pathology, while invasion of the lung may resemble a bronchopneumonia.

The diagnosis depends upon finding numerous small sulphur-like granules in the sputum, composed of thin mycelial filaments. Treatment should be instituted as early as possible by administration of large doses of potassium iodide, the end result being fairly good in most cases.

MONILIASIS

The first case of monilial infection of the bronchopulmonary system to be observed in this country was reported by Boggs and Pincoffs in Baltimore in 1915. Since then cases have been found in other parts of the country by different observers. Castellani found the disease fairly common in India and other tropical countries.

The infection may run a mild course, in which the general condition of the patient remains good, with little fever and few chest symptoms, the clinical picture resembling a chronic bronchitis.

These cases frequently clear up without complications. But there is another more serious type which runs a malignant course. The patient loses weight, has a cough with hemoptosis. The sputum frequently has a nauseating foul odor which is helpful in making a diagnosis. The physical signs and character of the disease resemble that of tuberculosis. Examination of the chest may show rales and patches of dullness. X-ray plates cannot differentiate between the two diseases because the changes are so similar.

A final diagnosis depends upon aspirating affected exudate with a bronchoscope and developing cultures upon specially prepared media.

Treatment, as in most of the mycotic diseases, consists in giving large doses of the iodides, but some of the cases fail to improve under any kind of medication and run a fatal course.

Although mycotic infections of the bronchopulmonary system might be termed relatively rare, enough such cases occur to be considered a bronchoscopic problem. In my opinion, many more of these infections could be discovered in obscure chest conditions, if more attention was given to them, and if there was closer cooperation between the internist and bronchoscopist.

While bronchoscopy is particularly valuable from a diagnostic standpoint in these cases, it has a field of usefulness for treatment as well. Some patients are greatly helped by aspiration and local medication as in the treatment of suppurative pulmonary infections. Local bronchoscopic measures combined with internal medication offer a fair prognosis in many cases.

Following I wish to briefly report a few cases, which have occurred in hospitals where I have been associated.. It is interesting to note the various types of infection found and the nature of involvement in the lower respiratory tract.

REPORT OF CASES

CASE 1.—S. D., age 36, single, dye worker, admitted to the Maryland State Santorium January 2, 1930; discharged August 2, 1930.

The past history was negative except for an attack of influenza in 1918. This case is interesting because it confirms earlier observations of the relationship of occupation to mycotic disease. This patient was a pigeon breeder and was in the habit of premasticating food, which he fed to the pigeons from his lips.

The patient gave a history of having had streaked sputum from 1919 to 1925. In 1925 he was treated at Perth Amboy, N. J., at the City Hospital, with a presumptive diagnosis of pulmonary tuberculosis. At that time sputum was persistently negative. He left there without much change in his condition, continued his infrequent slight streaking, and then in August, 1928, for the first time, began coughing sufficiently to cause annoyance. About six months previous to admission he began feeling unwell, had night sweats and chills, lost considerable strength and weight, and had a marked increase in the amount of his cough and expectoration. The patient reported to Johns Hopkins Hospital Dispensary and there an examination of sputum revealed numerous *aspergilli glaucus*. A diagnosis of aspergillosis of the lung was made and the patient was sent to the Maryland State Sanatorium for treatment of that condition.

On admission there was found, by x-ray, on the right side, a dense clouding of the right apex, slight fibrosis, first interspace; heavy roots at the base. The left lung showed clouding at the apex.

The chest examination was negative excepting for prolongation of expiration with WBY over the right apex, front and back.

The sputum findings were verified here at the institution, and the patient was again questioned in an attempt to explain the source of infection, at which time he stated that he had been a pigeon fancier for years, had premasticated the food, and had fed his pigeons from mouth to beak. The presumptive source of the aspergillus was the pigeon feed.

This patient had a perfectly normal temperature and normal pulse throughout his stay. He was treated with intravenous postassium iodide with marked success and left the institution seven months after admission in a markedly improved state of health with practically no cough and no expectoration, and greatly improved in strength.

Case 2.—C. T., aged 37, was admitted to the University Hospital December 7, 1923; died December 20, 1923.

Diagnosis: Actinomycosis.

The patient was sent into the hospital from the House of Correction, complaining of painful neck, sore throat and hoarseness. Examination showed dense, brawny thickening right side of neck extending from the hyoid bone to the clavicle; several openings which led into the mass, and discharging small masses resembling softer portions of actinomycosis. The patient has had a great deal of nausea and vomiting, consisting principally of bloody froth and mucus, which came from the larynx. He had a distressing cough and has had several attacks of marked dyspnea. Organisms expressed from the fistula in the neck were reported to be mycelium of actinomycosis.

The examination of the larynx showed a fistulous opening in the larynx which apparently communicated with the mass in the neck. It was a question whether the lesion originally started in the larynx and pharynx, or if the fistula was secondary to the lump in the neck. In any event the bronchial symptoms were secondary to the infection above.

Case 3.—S. H., age 23, farmer, admitted to University Hospital October 1, 1935; discharged October 23, 1935, condition improved.

Diagnosis: Bronchopulmonary moniliasis; chronic bronchitis.

The patient was sent into Chest Clinic at the hospital with a history of having had cough and expectoration of blood over a period of four months. He claims he had coughed a great deal during this time but did not expectorate any blood until June, 1935, at which time he coughed up over a cupful of blood and some material that looked like pus. Since that time he has had frequent attacks of coughing followed by blood streaked sputum. Has had no chills, no night sweats and suffered practically no pain. Mycotic infection was suspected and he was referred to the Bronchoscopic Clinic for study. The culture made from exudate aspirated revealed fungus infection of *monilia albus*. After treatment at the hospital the patient seemed improved.

Case 4.—J. M., aged 58. The patient had been treated in the Chest Clinic in the Dispensary for several weeks, complaining of pains in his chest, cough which was not productive. No chills, no fever. Patient was referred to Bronchoscopic Clinic and sputum showed mycotic infection of pulmonary system.

Bronchoscopy was performed, exudate aspirated and sent to laboratory for culture. Examination revealed aspergillosis of the niger type.

CASE 5.—Mrs. A. E. F., housewife, aged 60, was admitted to the Maryland State Sanatorium, August 31, 1931; discharged September 28, 1931.

The past history was negative except for whooping cough when a child and influenza and pneumonia in Ocober, 1930. Married at the age of 20. No children.

On admission the patient gave a history of having had influenza with bronchopneumonia on the right side, in October, 1930. Since that time she had been well. From October, 1930, until date of admission she had complained of some cough and slight expectoration, malaise, asthenia and loss of appetite.

X-ray on admission reads: Right lung, fibrous infiltration from the apex to R. 2; dense infiltration from R. 4 to base. Left lung, fibrous infiltration from the clavicle to R. 3, heavy roots at the base.

Impression at that time was that the condition was probably nontuberculous. Her temperature and pulse were entirely within the limits of normal. Her sputum was examined repeatedly for tubercle bacilli and consistently found to be negative for tubercle bacilli, and repeatedly there were found in fresh specimens fungus-like organisms which were identified by Dr. E. Reese Wilkens at the Sanatorium and the Laboratory of the Maryland State Department of Health as *"Coccidoides Immitis."*

Infection with this fungus has been known in the San Joaquin Valley of California and on closer questioning a history was elicited from this patient that she had visited this section of California four years previous to her admission to the Santorium.

This case was definitely considered as "Coccidoides Immitis" disease of the lung, and this patient was advised to leave the Sanatorium and obtain treatment at her home.

CASE 6.—Miss J. W., graduate nurse, age 28, single, was admitted to the hospital August 24, 1931, and discharged October 4, 1931.

She gave a history of having had chorea and articular rheumatism at the age of eleven; had tonsillectomy at the age of 21; bronchopneumonia 18 months previous to admission.

Her present illness began with grippe and bronchopneumonia in the spring of 1930. Following the grippal attack she had attacks of chills and fever periodically and after carrying the fever for a few days she would cough up a few ounces of sputum and then experience relief from her chills and fever.

Recommendation was made that she have vaccine therapy with a vaccine prepared from secretions removed directly from the focus of infection in the right bronchus.

An area of ulceration, from which she had repeated hemoptyses, was found.

Bacteriologic examination of specimen removed through the bronchoscope from the infected area showed the presence of *Aspergilli Conidia.*

Pneumothorax was attempted for a while to control the hemorrhages, with no success.

Examinations of this patient during her stay at the Sanatorium were almost entirely negative, except for the presence of a few bronchial rales in the interscapular region on the right side.

X-ray revealed heavy roots at both bases.

Sputum examinations were repeatedly negative for tubercle bacilli.

Diagnosis was made of aspergillus ulceration of the right bronchus and she was advised to leave the Sanatorium and secure treatment elsewhere.

CASE 7.—Miss L. B. W., housekeeper, age 20, single, was admitted January 6, 1932; discharged July 30, 1933.

She gave a history of having had scarlet fever, whooping cough and measles as a child. Menses began at age 14, but have been irregular as to time ever since. Had pleurisy in 1929 on the right side and pneumonia in 1928.

On admission she gave a history of having cough and expectoration for the past five years, coughing up blood in small amounts at intervals during these five years. Recently she had lost weight and strength, felt much worse than usual, reported for examination and was advised to come to the Sanatorium to take the cure for tuberculosis.

X-ray on admission indicated, on the right side, a fibrous infiltration to R. 3, with many heavy roots at the base. Left side: infiltration to R. 2, with fibrosis from R. 2 to R. 4, with heavy roots at the base.

Physical examination of her chest showed an impairment of percussion note on the right side from the apex to R. 2 in the front and from the apex to 4 V. S. in the back, with sonorous and sibilant rales heard over this area. Left side was negative.

Throughout her stay numerous sputum examinations were taken, and consistently they were negative for tubercle bacilli. Her temperature and pulse were normal throughout her stay, and she seemed to be doing very well until the latter part of June, 1933, when she expectorated several small clots of blood. In the middle of July she again expectorated a little blood, and at the end of that month she left the Sanatorium against our advice, with her diagnosis still very uncertain.

On November 13, 1933, she was again admitted to the Sanatorium, with a history of having had a rather large hemorrhage very shortly after her discharge from the institution, and after that hemorrhage she had streaked repeatedly until the time of her second admission.

On her second admission a peculiar condition of her finger nails was manifested. She was found to have fungus disease of all of her finger nails on both hands.

With a history of a large hemorrhage, it was thought well to attempt a bilateral pneumothorax, but a satisfactory collapse was never obtained and that was very quickly discontinued.

She was bronchoscoped twice at this institution and a specimen obtained from her bronchi contained very heavy growth of *Aspergillus Fumigatus*, and a diagnosis of aspergillus disease of the lung was made.

She was advised to leave the Sanatorium as a nontuberculous case.

CASE 8.—E. J., colored girl, age 15, was admitted October 6, 1928, to Tuberculosis Service (City Hospital). Died October 28, 1928.

The onset of the present illness was four or five months prior to admission, with pain and burning sensation in upper right quadrant. She contracted a cough which progressed and became only slightly productive. Hemoptysis on one occasion. A swelling developed in the upper right quadrant and later a swelling with a draining sinus under the costal margin on the right posteriorly. Yellow granules extruded with pus.

Examination revealed a draining sinus below the right scapula. The lungs revealed basilar infiltration on both sides with consolidation of the left lower lobe. Large indurated mass was felt in the left upper quadrant.

X-ray of the chest showed soft infiltration of the right lower lobe with consolidation of the left lower lobe.

Sputum revealed sulphur granules as well as discharge from draining sinuses. Hb., 40 per cent; W. B. C., 24,000; R. B. C., 3,700; P. M. N., 74 per cent. Wassermann negative.

Her course in the hospital was progressively downhill to a fatal termination. The patient had a septic type of fever chart.

Diagnosis: Actinomycosis, involving abdominal and chest walls, and lungs. Confirmed by autopsy.

CASE 9.—J. G., white man, age 43, admitted July 13, 1929, to Tuberculosis Service (City Hospital). Died December 7, 1929.

The complaint was of a productive cough, with pain in left chest and back since April, 1929. In June he noticed a swelling in the left chest with severe pain. Night sweats since May, 1929. Constant loss of weight with weakness.

Physical examination showed an emaciated, anemic white male with marked clubbing of fingers. Chest revealed infiltration throughout left lung with cavitation. The liver was enlarged. There was a fluctuant indurated mass in the left chest between the second and fifth interspace.

The laboratory findings were: Sputum negative for tubercle bacilli. Hb., 70 per cent; R. B. C., 3.48; W. B. C., 14,800; P. M. N., 76 per cent. Wassermann negative.

The x-ray examination showed a tuberculous infiltration throughout the left chest with cavitation; slight infiltration of the right apex.

Actinomycotic granules were recovered from sputum and pus from chest wall abscess.

The patient ran a continuous elevated temperature between 100 and 101 degrees. Abscess on chest and wall broke down and discharged through multiple sinus tracts. Hemoptysis occurred on one occasion. Course progressively downhill to fatal termination.

Diagnosis: Actinomycosis involving lungs and chest wall. Metastatic abscess to right thigh. Confirmed by autopsy.

CASE 10.—E. E., white man, age 63. Admitted August 17, 1932, to Tuberculosis Service (City Hospital). Died October 9, 1932.

The patient complained of pain in the left lower chest; cough and expectoration. Sputum was heavy and purulent in character.

Physical examination showed biapical fibrosis with evidence of fluid or thickened pleura at left base.

The laboratory findings were: Sputum and pleural fluid negative for tubercle bacilli. Blastomycosis was cultured from sputum. Hb., 68 per cent; R. B. C., 3.43; W. B. C., 5,700; P. M. N., 74 per cent. Wassermann negative

A small amount of fluid was obtained on several occasions from the left chest. Sputum culture revealed *blastomycosis.* Started on K. I. and symptoms became much more marked. Course became progressively downhill to fatal termination.

Impression.—1. Pulmonary gangrene; 2. Blastomycosis; 3. Fibroid pulmonary tuberculosis, apical.

CASE 11.—B. A., colored man, age 25, was admitted November 10, 1932, to Tuberculosis Service (City Hospital). Died September 21, 1933.

The patient was perfectly well until April, 1932, when he began to have a cough. Cough became productive two months later. Increasing dyspnea and progressive loss of weight.

The physical examination revealed widespread changes, more marked in the right lung, with steady progression leading to signs of consolidation in both lungs.

The laboratory findings were: Urine negative. Sputum negative for tubercle bacilli. Fungus growth reported on two occasions (penicillium). Blood: Hb., 75 per cent; R. B. C., 4,2000,000; W. B. C., 10,400; polys, 65 per cent; S. L., 30 per cent. Wassermann negative.

The course in hospital was progressively downhill with loss of weight and increasing dyspnea. Patient was bronchoscoped to obtain secretion directly from the lung, which was cultured and also showed penicillium fungus growth.

X-ray examination in November, 1932, showed extensive soft and fibroid infiltration on both sides. In April, 1933, there was an extensive increase in infiltration in both lungs, possible pneumoconiosis.

SUMMARY

1. Mycotic diseases affecting the lower respiratory tract can be only definitely diagnosed by bronchoscopic examination and study. Aspiration of exudate in the bronchi, uncontaminated by infections found in the mouth and pharynx, combined with bacteriologic study, is the only certain method of recognizing these maladies.

2. Such conditions demonstrate the practical advantages to be obtained by consultation with bronchoscopists in the diagnosis and treatment of obscure diseases of the pulmonary system.

3. While fungus infections are most frequently found in tropical countries, sporadic cases occur in sufficient numbers in other parts of the United States to warrant the attention of all bronchoscopists.

4. Most of these cases are sent into tuberculosis sanatoria with suspected tuberculosis, and the accurate diagnosis demonstrates the value of a bronchoscopic clinic in connection with every tuberculosis hospital, a subject I called attention to several years ago.

5. It is important that a positive diagnosis be made in these cases because the treatment of tuberculosis is absolutely different from that of fungus diseases affecting the lung. In mycotic diseases, iodides offer the most hopeful method of cure, a drug never to be used in pulmonary tuberculosis.

6. Bronchoscopy is also of value in treating these cases. Aspiration, with local medication, similar to methods employed in treating suppurative pulmonary infections, helps many of these patients.

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XCVII

LARYNGITIS AND TRACHEOBRONCHITIS: REFERENCE TO NONDIPHTHERITIC INFECTIONS*

T. R. Gittins, M.D.

Sioux City

Three years ago this subject of laryngotracheobronchitis of nondiphtheritic origin in children was thoroughly and scientifically presented before this group by Dr. Lyman Richards. Nothing I can say today can do more than emphasize some of the points he made at that time. I think, however, that this subject of laryngotracheobronchitis of nondiphtheritic origin is of sufficient importance to merit repetition. The very high mortality rate among infants and young children, even when the condition is recognized early and treated as best it can be, warrants some effort to acquaint the rank and file of otolaryngologists, pediatricians and general physicians with the facts. My excuse for bringing up this subject before a group thoroughly familiar with what I will have to say is to urge more missionary work among those who are seeing a great number of these cases and are not familiar with the special problems.

It is not enough that the medical profession be counseled that the Klebs-Loeffler organism is blamed for many conditions which it does not produce—it is of distinct importance that it be encouraged to realize that the relief of serious dyspnea with its accompaniment of dehydration, cardiac exhaustion, atelectasis or bronchopneumonia demands very special treatment by men thoroughly trained and equipped to do endoscopic work. The otolaryngologist doing casual endoscopy with insufficient equipment and training may get into more trouble than the pediatrician or general practitioner, because he will almost surely see some of the serious cases. How many cases diagnosed and treated as bronchopneumonia are really atelectasis secondary to mechanical obstruction in the trachea and bronchi, we do not know, of course, but it is reasonable to believe that a great many of these patients die because of lack of ventilation of their lower air passages. It has always seemed remarkable to me how much

*Read before the nineteenth annual meeting of the American Bronchoscopic Society, Detroit, May 27, 1936.

thick discharge one finds at tracheotomy, even when there seemed to be symptomatic evidence of only laryngeal involvement. Naturally, the pediatrician or general physician, as a rule, does not realize this possibility, and unless there is evidence of marked dyspnea of the upper respiratory type, he usually feels no need of help from the laryngologist. It is common knowledge among men doing bronchoscopy that the higher the obstruction is in the respiratory tract the more external evidence there is of dyspnea; and conversely, the lower the obstruction, the less evidence of dyspnea as represented by supra- and infra-sternal retraction, or cyanosis. But the general medical men usually do not realize this fact. Again, the cyanosis of rather sudden laryngeal obstruction is dramatic and self evident. It spells an emergency to anyone in charge of the patient and measures are immediately taken to relieve it or to call for help. On the other hand, the pale cyanosis, restlessness, high fever, dehydration and weak pulse, so consistent with obstruction of the smaller bronchial divisions with possible atelectasis, are not dramatic and certainly are not recognized as obstructive symptoms by the rank and file of medical men. The symptoms are considered secondary to toxemia because of the severity of the pneumonic infection.

We in this specialty must wonder and speculate in how many of the great number of young adults with bronchiectasis, the primary insult to the bronchial and lung tissue came about because there was insufficient drainage of the lower air passages during an attack of laryngo-tracheo-bronchitis at an age when the small larynx with its subglottic swelling and the lack of cough combined to prevent proper drainage. Perusal of the literature of the last few years indicates that many internists are becoming more conscious of the probable part played by infant bronchial infections in the production of bronchiectasis. The damage done to the bronchial mucosa and submucosa by infection, with the resultant fibrosis and dilatation of the bronchi seems to explain the pathology of the condition so commonly seen during bronchoscopic examination of clinical bronchiectasis. The squamous type of epithelium replaces the columnar ciliated type. Naturally drainage is interfered with, just as it is in the sinuses when the ciliated mucosa is damaged by infection.

In 1920, we realized for the first time that there were cases of serious laryngitis and tracheobronchitis in young children in which the onset and clinical picture simulated diphtheria, but in which antitoxin gave no relief and mechanical obstruction below the cannula was decidedly more troublesome than in true diphtheria.

REPORT OF CASE

CASE 1.—Our first patient, a child of nineteen months, was seen because of croupy cry, cough and spells of cyanosis and increasing dyspnea. Examination revealed negative tonsils and pharynx. There was moderate edema of the arytenoids and upper larynx, the true cords were only slightly swollen, but there was marked subglottic swelling which was covered by thin gray exudate. Tracheotomy was done. In the trachea there was a large amount of thick brownish sticky secretion. Semisolid masses of this same viscid material were coughed out of the tracheotomy wound. Diphtheria antitoxin was given intravenously. Dyspnea was greatly relieved after tracheotomy for several hours, but there were periods of cyanosis and restlessness. Twenty-four hours after tracheotomy, breathing was rapid and shallow, pulse weak and thready, and temperature 106. Breath sounds were deficient over both lungs, but there were no other signs indicating obstruction below the cannula. Laboratory reported cultures negative for diphtheria bacilli and positive for streptococci. A bronchoscope was passed through the tracheotomy wound. Large plugs of thick brownish tenacious material were removed from the main bronchi. There was some relief of dyspnea, but the child died within a few hours, apparently of cardiac exhaustion.

At autopsy the mucosa of the larynx, trachea and large bronchi were covered by a thin gray exudate. There was sticky discharge in the trachea and large bronchi. The smaller bronchi and bronchioles were practically occluded by this same viscid semisolid discharge. These postmortem findings helped to confirm the impression held before death that the serious symptoms were probably due more to mechanical obstruction than to toxemia. The semisolid masses of material from the smaller bronchi were made up of fibrin, leucocytes, some epithelial cells and organisms which were apparently streptococci.

In 1920 and 1921 three cases of this particular type were encountered. All died, even though tracheotomy was done early, antitoxin was given early, and efforts were made to relieve the mechanical obstruction of the trachea and bronchi below the canula. As similar conditions continued to appear, we came to realize more and more that the mechanical obstruction below the canula was of much greater importance than the causative organism or the resulting toxemia. Antitoxin was still given in large doses early, because we wanted to play safe if the disease proved to be diphtheria; and felt possibly the foreign protein reaction would be of benefit in the nondiphtheritic infections. In the early cases suction through the tracheal canula was used only when there were signs of recurring dyspnea. Many times nothing would be obtained by suction, and the rapid respiration, restlessness and pale cyanosis were attributed to the severity of the infection. Inspection with the bronchoscope and removal of semisolid plugs from the main bronchi with quick relief of all the serious symptoms, made us realize that something more must be done routinely to relieve mechanical obstruction. Bronchoscopic removal of these plugs was a life saving emergency procedure just as tracheotomy

had been earlier in the treatment, but it was attended by a certain amount of trauma and shock to the very sick infant. Various solutions dropped into the cannula at regular intervals, followed by suction, regardless of the apparent absence of dyspnea at the time, was rewarded by removal of thick discharge and often sticky plugs by the catheter or the cough that was produced by manipulation. We realized that this constant routine plumbing carried many seriously sick patients along without recourse to bronchoscopy.

Adrenalin, 1 to 10,000, or normal saline solution, has been used by us, but undoubtedly other solutions do just as well. Liquifying the discharge or keeping solid masses or plugs from forming is the goal in the use of any of these solutions. As we began to see decided improvement in our results by using solutions and suction routinely, we also made strenuous efforts to keep up a higher fluid intake than had been done previously. Fluids were given subcutaneously and intravenously as a routine procedure, and again it seemed that some advance was being made in the treatment of these very serious conditions.

For several years nothing new was added in the treatment until the oxygen tent was developed. Certainly in some of the milder cases it has seemed that the addition of oxygen has probably carried some patients along without the necessity of tracheotomy.

Regardless of the fact that we recognized these nondiphtheritic infections of the tracheobronchial tree in 1920, and have tried consistently to give these patients the benefit of early and constant care, and have used all methods of treatment suggested by perusal of the literature and by our own experiences, our mortality rate has remained consistently higher than when dealing with true diphtheria. Our mortality in tracheotomy cases during these past sixteen years has been 35 per cent in true diphtheria and 60 per cent in nondiphtheritic infections. A study of the literature reveals that many authors have reported an even higher mortality rate than this, but in many instances only a few cases were used for statistical study or small groups of a fulminating type only were under consideration.

In 1926, before the national meeting of the American Laryngological, Rhinological and Otological Society in Montreal, I discussed this subject of nondiphtheritic laryngo-tracheo-bronchitis. Fourteen cases were reported which had been encountered during the six years since we had recognized the clinical entity. It was mentioned that these cases were not reported with the thought that they were rare but rather because we felt that such conditions were relatively com-

mon, but were being diagnosed and treated as diphtheria. Ten of the fourteen cases had tracheotomy. Four recovered and six died, a mortality rate of 60 per cent.

A fairly complete review of the literature from 1914 was given. Fourteen references appeared in the bibliography. Personal communications from the commissioners of public health in ten of our larger cities were abstracted briefly. From one city it was stated by the commissioner that during measles or influenza outbreaks from 10 to 20 per cent of supposed cases of laryngeal diphtheria showed cultures negative for diphtheria bacilli and positive for streptococci. This writer went on to say that in his city 3 per cent of all the well children showed cultures positive for diphtheria-like bacilli which the laboratory calls diphtheria bacilli. Many of these children were Schick negative.

In this same paper I included abstracts of articles by Drs. Jackson, Clerf, Baum and the late Dr. Lynch, besides several others, to emphasize the comparative frequency and severity of these non-diphtheritic infections, and the high mortality rate, even when the real nature of the disease was recognized early. Dr. Lynch and Dr. Clerf kindly discussed this paper and added valuable suggestions as to the prevalence of the disease and its diagnosis and treatment.

Again, in 1932, before the American Laryngological Society in Atlantic City, I discussed this subject under the title "Laryngitis and Tracheobronchitis in Children—Special Reference to Nondiphtheritic Infection." At this time the case records of 24 patients encountered since 1920 in the private practices of my associate, Dr. Naftzger, and myself were abstracted. In fifteen, tracheotomy was done. Seven of these recovered and eight died, a mortality of 53 per cent. Nothing new was suggested in the treatment that had not been mentioned in 1926. We did try to emphasize, however, the importance of the routine use of weak adrenalin, saline or some other liquifying solution in the tracheotomy cannula every twenty minutes, or less if necessary, followed by suction, regardless of the severity of the dyspnea. With this simple treatment we felt quite sure that we had prevented in many instances the formation of the thick sticky discharge and plugs, and practically eliminated the need for bronchoscopy in removing these obstructions. We also emphasized the importance of a high fluid intake and the oxygen tent, especially in carrying along the milder cases without tracheotomy.

In this paper, in 1932, some thirty-five references from the English literature were listed with statistics of experiences by the

several authors with from one to over twenty cases. Because of the length of the bibliography, the individual references were not abstracted. For the most part in these articles on the subject there was the notation that nondiphtheritic infections of the larynx, trachea and bronchi were very rare. Almost invariably the authors called attention to the lack of reference in the literature to similar cases. Quite often only two or three references appeared in the bibliography and these referred to articles in local, state and sectional journals. One writer described a single case with the symptoms and clinical course of nondiphtheritic infection as diphtheria, but hastened to add that the case was so typical of diphtheria that no cultures were taken. Three papers from one small locality were found reporting from one to three cases. The bibliography of each report included only one reference to the other two articles. Hasty scanning of a few journals often brings forth the comment that the condition under discussion must be rare and the lierature scanty. Within the last year I have received a reprint of a paper on nondiphtheritic laryngitis and tracheobronchitis from one of our larger Southern cities. In discussing the paper, one otolaryngologist made the statement that in the presence of such a clinical picture as described by the essayist, one must assume that the condition must be diphtheria. The discussor mentioned that he could not personally recall a case of nondiphtheritic laryngotracheal involvement of any serious nature, but that all such anatomic extensions downward from the larynx were diphtheritic in origin. These comments on the literature are given to emphasize that there still is some need for repetition in calling attention to the probable frequency, and lack of recognition of these nondiphtheritic infections, even before men of our specialty.

At the present time our records show that since 1920 we have cared for thirty-two cases of these nondiphtheritic infections of sufficient severity to demand hospital observation and treatment. Many mild cases are not included in the series. Of these thirty-two cases, twenty had tracheotomy with subsequent treatment by suction and installation of fluids into the cannula and high fluid intake intravenously or subcutaneously as part of their postoperative care. Of these twenty tracheotomized cases, eight recovered and twelve died, a mortality rate of 60 per cent. After having recognized the nature of these nondiphtheritic infections for sixteen years and having tried every known method of treatment, our mortality rate is just as high as it was when a smaller number of cases were reported in 1926.

During the past year, for the first time I encountered an adult patient whose clinical course and physical findings simulated that

which we have been seeing in children for many years, especially in relation to the obstruction due to subglottic swelling in the larynx.

REPORT OF A CASE

CASE 2.—Mrs. E., age 29, a large woman of some 190 pounds was seen because of gradually increasing dyspnea, which had been preceded by hoarseness and some cough for five or six days. There had been spasms of coughing during which thick discharge and some plugs had been expelled. Cyanosis and difficult breathing had been relieved by these coughing attacks.

The patient, when first seen, was sitting up in a semistuporous condition with definite cyanosis and fairly marked dyspnea with supra- and infrasternal retraction. There was no acute inflammation of the tonsils, pharynx or in the nose. The true cords were only slightly thickened and pink. There was some edema over the arytenoids. Subglottic swelling was very marked on each side and seemed to practically obstruct the lumen of the larynx. There was a very thin exudate over this subglottic region with crusts in the interarytenoid space. Tracheotomy was considered and seemed indicated, but it seemed safer to transport the patient to the hospital first. In the hospital she was placed in an oxygen tent and saline solution was given intravenously. Temperature was 100 ; WBC 15,000. After a short time in the oxygen tent there was less dyspnea and tracheotomy was postponed. Internist reported rales over both bases—heart not especially embarrassed—area of bronchial breathing posterior left side, possibly due to atelectasis. Within twenty-four hours the edema of the arytenoids had receded, the subglottic swelling on the right side had reduced in size and dyspnea was less marked. Voice was very weak and hoarse because the true cords could not approximate, due to the subglottic swelling. Some plugs and crusts were coughed out occasionally. Laboratory reported from the culture—no diphtheria bacilli, many staphylococci and streptococci. Oxygen and free fluid intake continued. Each day there was gradual reduction of the subglottic swelling, less hoarseness and cough, less fever and clearing of chest. Discharged on the twelfth day in good condition.

Possibly adults with laryngitis and tracheitis may often have this marked subglottic swelling, but it is the first time I have encountered this particular physical finding which is so common in the young child.

In 1926, I called attention to the bacteriologic study of cultures from a small child with serious dyspnea, and the mother of the child who had been suffering from a mild acute laryngitis for several days before the child became ill. Streptococci isolated from the throat of the mother and larynx of the child were carried through the laboratory carefully on various types of media. The organisms proved to be identical in appearance and cultural characteristics under all circumstances.

We feel that many of the serious cases of laryngitis and tracheitis in infants and young children, are caused by contact infections from older children or adults who are suffering from comparatively mild

and ambulatory types of respiratory infections. Again I repeat for emphasis, as most writers on this subject do, it is the mechanical obstruction of the air passages of the infant and young child which is of more serious import than the type or virulence of the organism encountered.

Just recently it was our misfortune to lose a child of twenty months, five weeks after tracheotomy. This child came to the hospital with a history of sudden dyspnea following possible inhalation of a foreign body. A kernel of corn was removed from a bronchus. Tracheotomy was done soon after, because of continued dyspnea. There was a history of mild respiratory infection in the patient before the inhalation of the foreign body. The clinical course after tracheotomy was typical of that in the infective cases of tracheobronchitis. The usual routine, installation of weak adrenalin and saline into the cannula followed by suction was used. The patient made satisfactory improvement and the cannula was left out part of the time on the eighth day. The high temperatures had disappeared and there was very little discharge removed by suction. The general condition of the patient was excellent. At this time the mother, who was in constant attendance, developed an acute pharyngitis. The next day the father, who had stayed with the baby at nights, developed an acute follicular tonsillitis. Within two or three days the child again developed high fever, rapid respiration and became more seriously sick than at any time since admittance. The pediatrician reported areas in both lungs which he felt indicated bronchopneumonia. Despite constant efforts to keep the air passages clear and to keep up a high fluid intake, the child's condition became gradually more serious and death occurred five weeks after inhalation of the foreign body. It seems highly possible that the sudden turn for the worse and the subsequent fatality can be traced to the contact infection with virulent organisms from the acute infections of the parents, just at the time when the child was overcoming his original infection.

SUMMARY

To conserve time and space some rambling statements concerning personal experiences with the clinical condition under consideration will be listed in outline form.

As in other writers' experiences, we have found this disease entity is of clinical importance only in children, and the seriousness varies in inverse proportion to the age.

Only one adult with the marked subglottic swelling so common in the children has been encountered.

In our practice the real fulminating types with quick fatalities have appeared in very young babies.

We have not encountered this clinical condition in epidemic form, as has been reported by several writers. In sixteen years we have seen thirty-two cases and they have usually appeared one at a time, entirely different from the incidence of diphtheria.

Usually these little patients have had no acute infection in the tonsils, pharynx, nose or sinuses of any severity. Often they were up and about, with mild respiratory symptoms until croupy cough, dyspnea and an emergency developed over night.

The only distinct bacteriologic finding has been the lack of the diphtheria bacilli. Streptococci have been found most often in cultures, but were often associated with staphylococci and pneumococci.

The characteristic pathology has consisted of subglottic swelling and thick discharge in the trachea and bronchi, which has tended to form in masses and plugs. Thin gray exduate over thickened mucosa rather than membrane has been usual. Edema has been of no importance.

Tracheotomy is considered essential in the serious cases; intubation may suffice in mild cases, but is a serious impediment to proper treatment of the tracheobronchitis which is of most importance after cannulation. These infections are usually of a descending type.

Routine use of weak adrenalin, saline solution or some other liquifying agent, at regular intervals, followed by suction, is of greatest importance in treatment. Using this routine treatment often prevents the formation of sticky discharge and plugs, thus often preventing return of dyspnea and making less necessary use of the bronchoscope.

Prevention of the formation of these plugs is the best way to prevent atelectasis and its serious consequences.

Milder cases often respond to simple measures, such as steam inhalations, oxygen tent and high fluid intake. Hospital treatment is of utmost importance even in milder cases.

In three of the cases considered in our statistics, infective tracheobronchitis with its typical clinical course followed inhalation of foreign bodies and their removal. It seems only reasonable that many children would be suffering from a respiratory infection at the time they inhaled a foreign body. Undoubtedly many of these patients are lost after bronchoscopy because proper efforts are not

made to insure good postoperative plumbing of the lower air passages.

Experience with this condition emphasizes that the diagnosis of serious dyspnea is the important thing, regardless of the cause. Bacteriologic and x-ray studies can often be made much more safely after tracheotomy.

Diphtheria antitoxin should be given early. If its specific action is not necessary, foreign protein reaction might be valuable. Our experience with vaccine therapy, so strongly recommended by some writers, has been too meager to warrant any statement.

So-called bronchopneumonia must commonly be atelectasis secondary to mechanical obstruction of the smaller bronchi. Atelectasis is probably 50 per cent more common in these nondiphtheritic infections than in true diphtheria.

Chronic bronchiectasis in older children and young adults is possibly often secondary to the acute infective tracheobronchitis of childhood. Possibly proper treatment of this acute disease in the younger years may eventually reduce the incidence of this miserable chronic condition of later years.

408 Davidson Bldg.

XCVIII

FLEXIBLE CANE-SHAPED TRACHEOTOMY TUBE*

R. M. LUKENS, M.D.

PHILADELPHIA

This instrument was designed to facilitate the introduction of a cane-shaped tracheotomy tube.

In general construction it is the reverse of the regular cane-shaped tracheotomy tube, the outer cannula being a curved tube and the inner canula being a long cane-shaped tube. The curved outer cannula permits the flexible cane-shaped inner cannula to pass around the tracheofistular angle, without damaging the tissues.

Fig. 1. Flexible cane-shaped tracheotomy tube.

The construction of the inner tubes (*A A* in the cut) provides flexibility of the straight portion of the cane-shaped tube so that it can pass through the curve of the outer cannula and straighten itself when emerging into the trachea. Two inner cannulæ are provided so that a clean tube can replace the soiled or blocked one as often as necessary. Furthermore, the inner cannulæ can be made any desirable length so that if necessary the inner cannula could reach almost to the carina.

1923 SPRUCE ST.

*Presented before the nineteenth annual meeting of the American Bronchoscopic Society, Detroit, May 27, 1936.

XCIX

ARTIFICIAL LARYNX, WITH PATIENT DEMONSTRATING ITS USE*

SAMUEL IGLAUER, M.D.

CINCINNATI

I wish to present a patient who had a laryngectomy performed about four years ago and, being a mechanic, he made his own artificial larynx. He gets out a new model about every six or eight months.

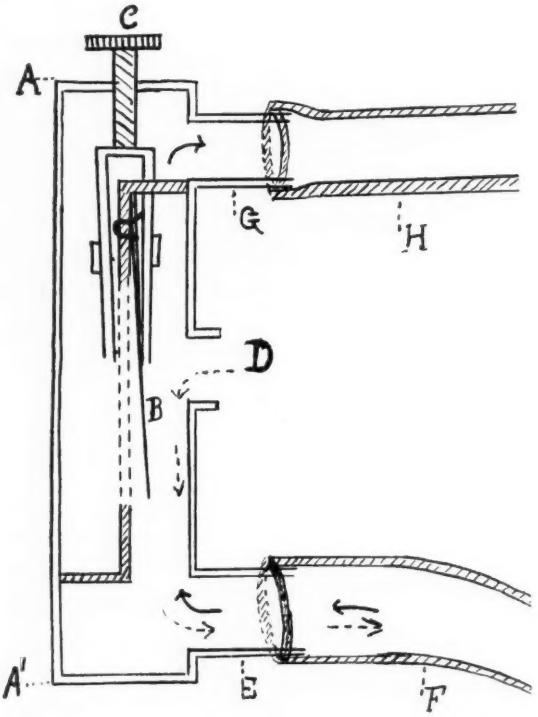

Fig. 1. Sectional view showing construction of artificial larynx.

*Presented before the nineteenth annual meeting of the American Bronchoscopic Society, Detroit, May 27, 1936.

This instrument is a modification of the usual artificial larynx on the market. It was made by the patient, Mr. Thomas Loring, who will demonstrate it.

The instrument (Fig. 1) consists of a brass tube (*A*-*A*') containing an adjustable brass reed (*B*), by the vibrations of which the fundamental tone is obtained. The reed works in an open slot (without any base), which gives it freedom of vibration. A special feature of the apparatus consists of the sliding contact (*C*) regulating the pitch of the reed. There is a hole (*D*) in the side of the outer tube, through which the patient controls the intake and exhaust of air. At the lower end of the instrument there is a hollow elbow (*E*) attached to the outer tube, and from this elbow a rubber tube (*F*) extends to the tracheal opening. At the upper end of the tube is another hollow elbow (*G*), to which a soft piece of rubber tubing (*H*) is attached. The latter is inserted in the mouth when speaking.

The advantages of this instrument lie in the use and seating of the metal reed, which will not stick in case saliva enters the apparatus, and, therefore, requires cleansing only at infrequent intervals. Furthermore, the sliding contact which the inventor has added enables the user to alter the fundamental tone of the instrument.

707 RACE ST.

C

FORCEPS FOR EXTRACTING A NAIL OR SCREW LODGED HEAD UPWARD FROM THE BRONCHUS*

CHEVALIER L. JACKSON, M.D.

PHILADELPHIA

I wish to present a forceps for dealing with the screw or nail lodged head-up in a bronchus. We are all familiar with this problem and know how difficult it is get the forceps blades over the head of the screw or nail.

One of the other forceps that has been most useful in dealing with this problem is the "double-claw" forceps. The double-claw forceps gives a very secure grasp, but it has four sharp points on the blades which do not readily slide over the head; secondly, these points do incur some risk of traumatism.

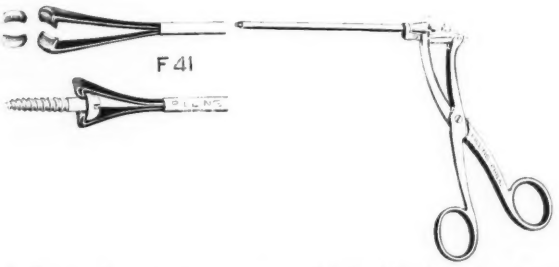

Fig. 1. Forceps for grasping screw or nail lodged head-up in a bronchus.

The forceps which I devised to meet this problem has blades which are curved in such a way as to facilitate the forceps sliding over the head of the screw. (Fig. 1.) This forceps is fairly stiff-springed, in order to dilate the bronchus to some extent, and thus obtain forceps spaces, but if it doesn't dilate the bronchus quite sufficiently, the very slightest advancing pressure tends to engage the forceps over the head of the screw rather than pushing it down, and once the head is grasped the grasp is very secure.

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*Presented before the nineteenth annual meeting of the American Bronchoscopic Society, Detroit, May 27, 1936.

CI

(1) A CHILD SIZE ESOPHAGO-GASTROSCOPE; (2) AN ADULT ESOPHAGO-GASTROSCOPE*

GABRIEL TUCKER, M.D.

PHILADELPHIA

The instruments which I wish to present are modifications of the present model of Chevalier Jackson's full lumen esophagoscope. In the performance of flexible tube gastroscopy I believe that it is the best practice to examine the esophagus and cardiac end of the stomach with an open tube, even though the routine x-ray studies have been negative for evidence of abnormality. We are justified in examining the esophagus by direct vision in order that we may be certain that there is no contraindication in the esophagus to the blind passage of the flexible gastroscope. The introduction in the adult of the 9 mm. esophagoscope that is ordinarily used may cause some

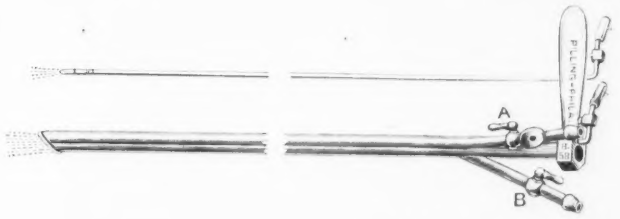

Fig. 1. Esophagogastroscope. Two sizes. Child's, 6 mm. by 53 cm.; adult's, 8 mm. full lumen by 58 cm. *A* combined thumb-valve suction release and stop-cock; *B* Stop-cock on insufflation tube.

little discomfort and soreness following the examination. In order to avoid this discomfort I therefore devised the esophagogastroscope which I wish to present.

1. *Child Size Esophagogastroscope* (Fig. 1).—This tube is 6 mm. full lumen by 53 cm. in length. It is provided with a special stop-cock and "suction release thumb valve" (Fig. 1*A*) combination which I believe to be original in this instrument. This tube will enable visualization of the entire esophagus in the

*Presented before the nineteenth annual meeting of the American Bronchoscopic Society, Detroit, May 27, 1936.

adult of the usual height and can be passed immediately prior to flexible tube gastroscopy without discomfort or soreness in the esophagus following its use. This 6 mm. full lumen by 53 cm. child size esophagogastroscope can be used quite satisfactorily for gastroscopy in a child. With it the left half of the child's stomach can be satisfactorily explored.

2. *The Adult Esophagogastroscope* (Fig. 1).—The second instrument I wish to present is a duplicate of the first, except that it is 8 mm. full lumen by 58 cm. With the adult size instrument it is possible to explore the greater portion of the cardiac end of the stomach of the adult of usual height. The 8 mm. full lumen size gives an ample field of vision, and the 58 cm. gives sufficient length of the instrument for deep exploration of the stomach, and through it may be used the usual 60 cm. forceps equipment. Dr. Harris P. Mosher points out the necessity for careful dilatation of the constricted area in the hiatal esophagus in so-called cardiospasm. In my experience the safest and most satisfactory way to dilate this area is to use the adult esophagogastroscope, dilating the stenosis with bougies in increasing sizes through it until the 8 mm. tube will enter the stomach. After inspection of the stenosed area the Tucker pneumatic dilator is passed through the 58 cm. esophagogastroscope, the tube withdrawn far enough to allow the dilating bag to remain in the hiatal esophagus; air pressure is then applied and the stenosed area dilated as desired. The 58 cm. tube is long enough to insure proper placement of the dilating bag, even in six and a half feet patients.

In presenting these esophagogastroscopes I desire to call attention to the "thumb valve suction release" which I presented at the 1930 meeting of the Section of Laryngology, Otology and Rhinology of the American Medical Association, and which Dr. Jackson has adopted as standard equipment on all his full lumen esophagoscopes. The combination of my "thumb valve suction release" and Jackson's stop-cock I believe to be new and very desirable in this type of instrument (Fig. 1 *A*). The thumb valve suction release facilitates interruption of suction and the cut off valve prevents the escape of air when the stomach is inflated through the gastroscope. Both these instruments are provided with the usual window plug for open tube gastroscopes, and during the past year have filled, in my hands, most satisfactorily an important place in the instrumentarium for examination of the esophagus and stomach in both the child and adult. I believe that they will add a great deal to the safety and comfort of the patient during these procedures.

326 South Nineteenth Street.

Clinical Notes

CII

SALIVARY GLAND TISSUE IN THE TONSILLAR FOSSA

JULIUS PROHOVNIK, M.D.

CHICAGO

The finding of salivary gland tissue in the tonsillar fossa is so infrequent that its occurrence should be recorded for statistical purposes. A review of the literature of the last ten years revealed only three cases recorded. H. Neumann[1] described the finding of aberrant salivary gland tissue above and behind the upper pole of the right tonsillar fossa. His patient was a female, 26 years of age. In this article Neumann states that he could find no reference in the literature as to a similar case, and that the case reported was the first in his experience. R. S. Tillotson[2] and Ernest Seydell[3] each reported finding salivary gland tissue in the tonsillar fossa following the removal of tonsils. The latter two author's cases were boys, aged 10 and 5 years respectively.

REPORT OF A CASE

CASE 1.—Mrs. I. R., age 46 years, white. Referred from the medical department to the department of otolaryngology for a tonsillectomy because of a neuritis.

Past history disclosed that patient had an abscess in her throat about twenty years ago. Examination of the patient's throat revealed small atrophic tonsils. There was a small oval perforation in the posterior pillar of the right tonsil. A tonsillectomy by the dissection method under local anesthesia was performed. After the right tonsil had been snared off, leaving a rather deep cavity, a yellowish gray tissue of soft consistency was found filling the infratonsillar fossa. The first thought that occurred was that perhaps there was more lymphoid tissue. However, a more careful examination disclosed that the tissue was definitely lobulated. A tentative diagnosis

*From the otolaryngologic service of Drs. Lewy and Morwitz, Mt. Sinai Hospital, Chicago.

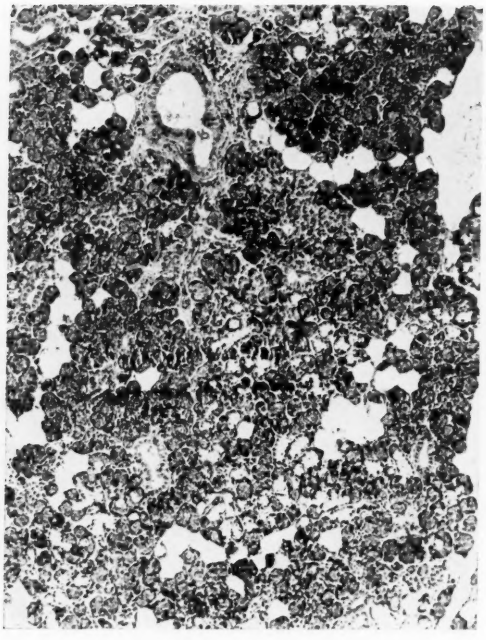

Fig. 1. Section showing submaxillary gland tissue. Low power.

of salivary gland tissue was made. A single lobule was then carefully dissected out for microscopic study.

Report of Pathologist (Dr. I. Davidsohn), Gross: Specimen consists of portions of the right and left tonsil. The portion of the left tonsil measures 13 x 9 x 4 mm. There are two separate portions from the right tonsil and from the adjacent tissue. There is also present a piece of tissue which was removed from the infratonsillar fossa on the right side. It measures 15 x 7 x 6 mm. The consistency is soft. The outside surface and section surface are yellowish gray.

Microscopic: Sections from the left tonsil show a thick squamous stratified epithelium on the surface and a great deal of granulation issue, and of new formed connective tissue in the underlying tissue. Muscle tissue and mucous glands are also present. Sections from the tissue removed and from the right tonsillar area show a great deal of connective tissue, fat tissue, a great deal of lymphoid tissue, and on the surface stratified, squamous epithelium. Under-

neath, lymph follicles are present. Islands of cartilage, some of which are calcified, are present, in the depth. The picture represents that of chronic inflammation. Sections from the tissue which was removed from the wall of the lower pole of the right tonsillar fossa show the structure of a salivary gland tissue without any histologic abnormalities. A subsequent report of this glandular tissue described the presence of both serous and mucous glands, the former predominating. The structure is characteristic of submaxillary gland tissue. Fig. 1.

Embryologically the presence of aberrant gland tissue in the tonsillar fossa could be explained on the basis of an anlage of salivary gland tissue becoming misplaced. It is also not likely that the presence of a peritonsillar abscess may have produced a perforation of the fibers of the superior pharyngeal constrictors muscles and facial planes which cover the floor of the tonsillar fossa, permitting the salivary gland tissue to evaginate through, because the intervening distance between the submaxillary gland and the tonsil is too great; furthermore this assumption in this case is strengthened by the fact that we had an oval perforation in the posterior pillar, which is not likely to have been the result of the above mentioned peritonsillar abscess.

After the complete removal of the tonsils the surgeon on finding any soft tissue in the tonsillar fossa should bear in mind the possible presence of salivary gland tissue and refrain from further surgical intervention.

3714 W. Roosevelt Rd.

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CIII

A NEW POSTOPERATIVE NASAL PACK FOR SUBMUCOUS RESECTION*

O. Jason Dixon, M.D.

Kansas City

Nasal splints have been generally accepted as proper means of holding the mucous flaps together following a submucous resection of the nasal septum. While these splints do perform this function, the amount of swelling which they undergo when saturated with water quite often produces an extreme and unnecessary amount of intranasal pressure. Also they have the disadvantage of completely occluding the nares and inhibiting a sufficient amount of drainage. As a substitute, I have been using strips of fine mesh sponge trimmed to a size which is a little larger than the normal nares and inserting with bayonet forceps. The side of the sponge adjacent to the nasal flap is slightly annointed with vaseline to prevent too firm an attachment to the point of incision. This vaseline coating prevents a firm attachment of granulation buds and permits an easier removal of the sponge strip. The sponge should be inserted moist after it has been sterilized. It should not be sterilized by heat or boiling, but preferably by such a solution as potassio-mercuric iodide (Parke-Davis), 1 to 5000 dilution, in which it should be left for twenty-four hours. The sponge is then removed to sterile normal saline solution, through which it is thoroughly washed several times and finally left in sterile distilled water until ready for use.

The forceps should grasp the sponge for its entire length, reaching to the tip, and should carry it well up into the nose, as it is too pliable to be pushed forward ahead of the forceps.

I find that the patient is much more comfortable with this type of soft packing, and also the sponge permits continuous seepage through it. It may be removed within twenty-four or forty-eight hours, as the surgeon desires. Following its removal there is very slight reaction on the part of the nasal mucous membranes, and the

*From the Department of Experimental Medicine of the University of Kansas School of Medicine.

turbinates, having been less irritated by pressure, are not so apt to swell and occlude the nares.

I was prompted to use this method of nasal packing after such satisfactory results on postmastoidectomy dressings.[1]

1210 PROFESSIONAL BUILDING.

REFERENCE

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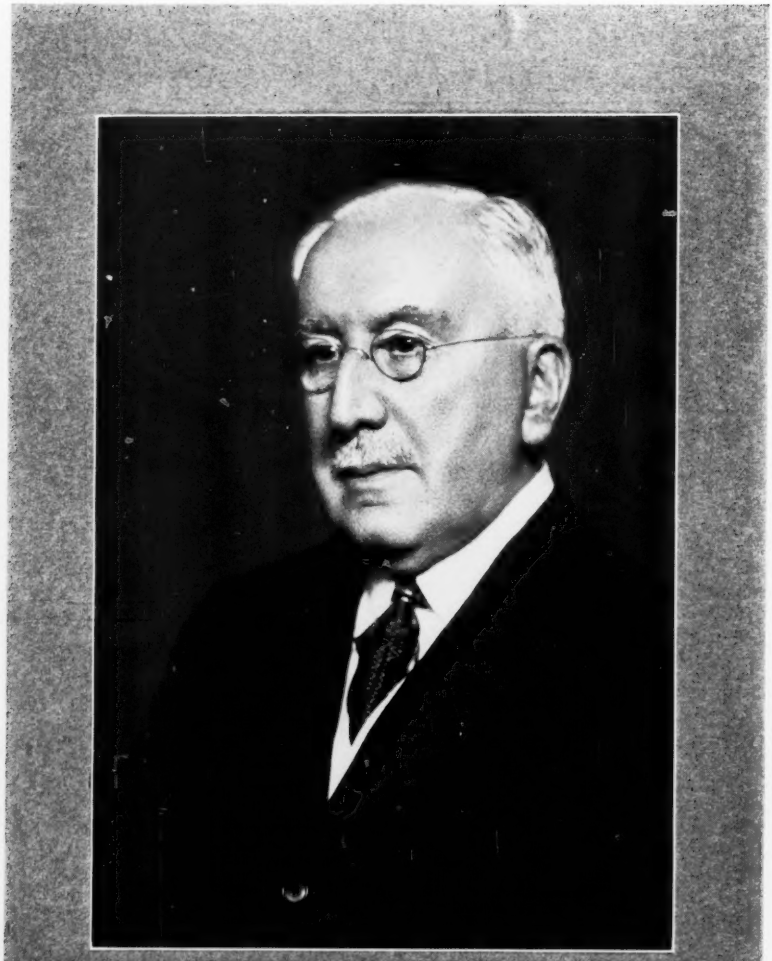

SIR CHARLES BALLANCE

SIR CHARLES BALLANCE

1856-1936

The death of Sir Charles Ballance on February 8, 1936, in his eightieth year, has deprived aural surgery in Great Britain of its chief pioneer. Sir Charles, who was born on August 30, 1856, was appointed Aural Surgeon to St. Thomas' Hospital more than fifty years ago.

He found then the treatment of suppuration in the middle ear in a most primitive condition, and with Sir William Macewean of Glasgow he led the way in establishing the proper surgical treatment of suppurative disease in the middle ear and of its complications.

He first advocated in England the use of epithelial Thiersch grafts after the radical mastoid operation.

He also held the appointment of Surgeon to St. Thomas' Hospital and to the National Hospital for Paralysis and Epilepsy in Queens Square, where his other surgical colleague was Sir Victor Horsley, and the physicians included such famous names in neurology as Gowers, Ferrier, Hughlings Jackson and Risien Russell.

He accomplished much original work in general and neurologic surgery, but never lost interest in aural surgery, and was always regarded as the leader by aural surgeons in Great Britain. He was devoted to research in surgical pathology and spent much time in experimental and laboratory research.

Even after his retirement he undertook, during the last five years, an elaborate research on the surgical treatment of facial palsy by means of nerve grafting, in conjunction with Dr. Arthur Duel of New York.

This work, which has established a new principle in the surgery of facial paralysis, was undertaken in a laboratory fitted up by Dr. Duel near his country home at Laurelwood in Duchess County, New York.

Sir Charles had many other activities, and was Chief Surgeon to the metropolitan police from 1912 to 1926.

During the Great War he was one of the Consulting Surgeons to the British Army, and in this capacity acted as Consultant to the base at Malta, where an enormous number of sick and wounded from the Eastern theaters of war were concentrated. For his services

there he received the Order of Companion of the Bath, and later was created Knight Commander of the Order of St. Michael and St. George.

He also held the offices of President of the Medical Society of London and Vice-President of the Royal College of Surgeons.

Sir Charles wrote much on neurologic surgery, the surgical treatment of aneurysm and on surgical pathology.

His principal contribution to aural surgery was two splendid volumes entitled the Surgery of the Temporal Bone, in which he gave a mature review of the whole subject of aural surgery on the basis of a wide knowledge and experience of surgery.

LIONEL COLLEDGE.

Books Received

Modern Treatment of Diseases of the Respiratory System.

A. Lisle Punch, M.B., M.R.C.P., Senior Physician, Royal Northern Hospital; Physician Brompton Hospital for Diseases of the Chest; and F. A. Knott, M.D., M.R.C.P., D.P.H., Director of Bacteriological Dept. and Lecturer in Bacteriology to Medical and Dental Schools, Guy's Hospital. Reader of Pathology, University of London. Cloth. Octavo of 295 pages with 96 plates and 31 text-figures. Philadelphia: P. Blakiston's Son & Co., Inc.: 1936. Price $5.00.

This is a compact manual dealing with the management of upper respiratory diseases from the common cold to bronchiectasis, pulmonary abscess, tuberculosis and tumors and aneurysms. It is written in the terse style commonly employed by English authors in these small texts. The material is well handled and the radiographs are excellent.

Diseases of the Air and Food Passages of Foreign Body Origin.

Chevalier Jackson, M.D., Sc.D., F.A.C.S., LL.D., Professor of Bronchoscopy and Esophagoscopy, Temple University, and Chevalier L. Jackson, A.B., M.D., M.Sc. (Med.), F.A.C.S., Professor of Clinical Bronchoscopy and Esophagoscopy, Temple University. 994 pages with 2000 illustrations including three plates in colors. Philadelphia and London: W. B. Saunders Company, 1936. Cloth, $12.50 net.

This is a veritable encyclopedia of endoscopy as it applies to foreign bodies.

The first section of the book (333 pages) is a text devoted to the entire field of this important specialty. The material is presented under ten heads: Etiology, prophylaxis, pathology, diagnosis, treatment, mechanical problems, extraction of foreign bodies from the esophagus, diagnosis and treatment of foreign bodies in the gastrointestinal tract, prognosis and instruments.

The second section of the book (636 pages) is devoted to nearly 3000 foreign body cases tabulated and illustrated in such a way that the thoughtful student may avail himself in detail of the experiences of these remarkable endoscopists.

The text is essentially an elaboration of the authors' previous publications, but the addition of the wealth of clinical material, each case with its problems and their solution clearly outlined, affords the reader an experience which so few personal practices can supply. Fur-

ther, it gives him an insight into the mental and mechanical attitude of the operator in a given case which no amount of didactic writing can convey.

It should be kept where the nurses and assistants can consult it.

Notice

THE AMERICAN BOARD OF OTOLARYNGOLOGY

An examination was held in New York City, September 25th and 26th, prior to the meeting of the American Academy of Ophthalmology and Otolaryngology. One hundred and twenty candidates were examined—of this number one hundred and three were certified.

An examination will be held in Philadelphia, June 7th and 8th, 1937, during the meeting of the American Medical Association, held in Atlantic City.

Prospective applicants for certificate should secure application blanks from the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, Nebraska.

W. P. WHERRY, M.D., Secretary.

H. P. MOSHER, M.D., President.

Abstracts of Current Articles

NOSE

Fifteen Years' Experience With Drainage Tubes After Antrostomy in Children.

Shea, John J. (Memphis), Arch. Otolaryng., 24:14 (July), 1936.

Conclusions presented in 1925, after five years' usage of these tubes, have withstood the test of time.

1. The end results of antral drainage in children compare favorably with those obtained in adults.

2. Radical surgical operation on the antrum is rarely necessary in children.

3. Suppurative or allergic processes will arrest the development of the sinuses, but by re-establishment of their normal ventilation and drainage they are pneumatized and usually regain their lost growth.

4. The use of ordinary rubber catheters, No. 12, 14 or 16 French, the size depending on the age of the patient, have been found satisfactory. A well placed tube remains in place, permits free drainage and is easily and painlessly removed; the self-retaining type of tube sacrifices caliber and is difficult to remove, extraction often being very painful.

The author describes his selection of cases, the operative procedure and the postoperative treatment, which includes autogenous vaccine therapy. Constant attention for one or two years is sometimes required in established sinusitis to effect normal functions.

TOBEY.

Ethmoidal Mucocele (De la mucocèle ethmoïdale).

Laskiewicz, A. (Poznan), Rev. de Lar., Ot., Rhin., 57:637 (June), 1936.

Seven of ten cases were enlargements of cystic middle turbinates invading the ethmoid; three projected up into the frontal and posterior ethmoid. All showed more or less orbital invasion with squint, lid swelling or diplopia, which ceased promptly after intranasal operation. The frontal and posterior ethmoid involvements required external surgery. Although the fluid was sterile in these cysts, the presence of osteoclasts and of proliferating periosteum suggests an inflammatory basis for the development of these growths.

FENTON.

PHARYNX.

The Clinical Significance of Compensatory Granular Pharyngitis.

Lillie, Harold I. (Rochester, Minn.), Arch. Otolaryng., 24:319 (Sept.), 1936.

A certain group of patients develop pharyngeal symptoms after tonsillectomy and regret having had the operation done. These cases of compensatory pharyngitis are analyzed. We are told how to suspect them pre-operatively, through the habitus, history and dietetic background of the patient, together with the appearance of the pharyngeal lymph nodes. The various methods of treatment are reviewed and none of them have been found as satisfactory as iodides in the form of ethyl di-iodobrassidate or syrup of hydriotic acid.

TOBEY.

Cerebellar Abscess of Nasopharyngeal Origin [Ascesso cerebellare d'origine nasofaringea (sindrome di Collet-Sicard).]

Antognoli, G. C. (Roma), Rev. Oto-Neuro-Oftal., 13:37-52 (Jan.-Feb.), 1936.

The author reports a case of lympho-epithelioid tumor located in the nasopharynx at the right peritubaric region, producing the Collet-Sicard syndrome.

Roentgen ray therapy reduced the tumor and also relieved the syndrome. Infection developed in the neoplasm producing a retrograde phlebitis, which through the submucous pharyngeal venous plexus extended into the internal jugular vein and the lateral sinus, causing an extensive necrosis of the occipital bone, at the right, which in turn produced meningo-encephalitis, subdural abscess and finally a right cerebellar abscess and terminated fatally.

The author calls attention to the variety of similar infections originating from the nasopharynx. He further discusses in detail the examination of postmortem material, to trace the route of the infection, and the histologic sections of the biopsy obtained from the peritubal tumor which was identified as "reticulosis lympho-epithelioid of Schmincke." Several photomicrographs and photographs accompany the article.

SCIARRETTA.

Acute Streptococcal Infections of the Throat.

Davis, Edward D. D. (London), Brit. Med. J., page 617, September 26, 1936.

In 1895 Felix Semon argued that all the various forms of acute septic inflammation of the throat known as a number of different conditions should be considered as varying degrees of virulence of one and the same pathologic process. There is a great variation in the clinical signs and symptoms depending upon the severity and type of infection, the resistance of the patient, and the situation of the

lesion. For clinical purposes two chief degrees of inflammation are recognized: 1. Tonsillitis, with considerable variation in the severity and in the extension of the inflammation and edema to the surrounding tissues; 2. A gangrenous inflammation, with a sloughing membranous exudate and toxemic and septicemic symptoms which often begin with acute tonsillitis, and is a later stage of the more severe types of the first group.

The etiology, treatment and complications are discussed. The most common causal organism is the streptococcus hemolyticus. Streptococci and pneumococci are often found together in the normal throat, and both these organisms will often be present in septic conditions of the throat, so that it may be impossible to say which is the causal organism and which is the secondary invader, or there may be a mixed infection. Streptococcal infections of the throat, even when they appear as an ordinary tonsillitis, demand careful treatment, and the complications are numerous, particularly in cases of a septicemic nature.

GOLDSMITH,
MCLEOD.

ESOPHAGUS, TRACHEA AND BRONCHI

The Hemodynamic and Respiratory Reflexes at Different Stages During Bronchoscopy and Esophagoscopy and Under Different Anesthetics (Les réflexes hémodynamiques et respiratoires aux divers moments de la bronchoscopie et de l'oesophagoscopie et dans différent conditions d'anesthésie générale et locale).

Bezzi, E., and Ciurle, L., Bronchoscopie, Oesophagoscopie et Gastroscopie (April), 1936. Page 156.

In this physiologic investigation, the authors recorded and studied arteriograms and pneumograms obtained in dogs during various endoscopic manipulations and under various anesthetics, and reached the following conclusions:

1. In the chloralized dog, respiratory and hemodynamic reflexes occur during the various stages of laryngobronchoscopy, more intense from the larynx, and although their intensity varies in different individuals they are constantly present.

2. These reflexes are present but diminished under morphin narcosis, are more marked under evipal anesthesia, and are reduced to a minimum under local anesthesia with 2 per cent pantocain.

3. Esophagoscopy produces much less marked reflexes under the various anesthetics and these occur only when the superior narrowing is passed and when the tube is withdrawn.

4. The respiratory and hemodynamic reflexes do not necessarily run parallel.

5. Trauma, even of the slightest, of the esophageal mucosa, exaggerates the reflexes and is a more important factor than the so-called "trigger zones."

6. Jackson's instruments produced less reflex response than those of Brünings.

7. Section of the superior laryngeal nerve abolished the reflexes from the larynx but not from the trachea. Section of the superior laryngeal nerves and the vagi abolished all reflexes.

8. The clinical deductions are that these reflexes do occur, are exaggerated by trauma, even of the slightest, and by the use of instruments of large caliber. General anesthesia is not as effective as local anesthesia in abolishing these reflexes, so that, even in young individuals, local anesthesia is to be advised, preferably 2 per cent pantocain. In esophagoscopy, anesthesia of the superior entrance is all that is necessary. The esophageal mucosa stands operative trauma better than the bronchial mucosa.

KLAWANS.

The Value of Bronchoscopy as an Aid in the Diagnosis and Treatment of Bronchial Carcinoma.

Ballon, David H., and Ballon, Harry C. (Montreal), J. Laryng. and Otol., 51: 499 (Aug.), 1936.

Diagnosis of bronchial carcinoma is often possible by bronchoscopy earlier than by other means. It is often possible to estimate operability.

Published results indicate that radium causes the disappearance of the endoscopically visible portion of the tumor. It is the authors' opinion that many tumors which have responded to treatment per bronchoscope were probably not true carcinomas.

Bronchoscopy in Hemoptysis.

Gerlings, P. G. (Amsterdam), J. Laryng. and Otol., 51:508 (Aug.), 1936.

The author recommends bronchoscopy particularly in cases of hemoptysis in which clinical examination is negative. Not only is diagnosis often possible, but effective treatment may be given.

Mucosal and Submucosal Cancers of the Esophagus (Cancers muqueux et sous-muqueux de l'oesophage).

Loza, Gonzalez, Bronchoscopie, Oesophagoscopie et Gastroscopie (April), 1936. Page 141.

Discussing esophageal cancer as it is met in his clinic in Argentine, the author notes that a high percentage of lesions are found in the middle third of the organ. He separates as a distinct entity sub-

mucosal cancers which give a distinct clinical, x-ray and endoscopic picture. Originating in the esophageal glands of the submucosa, these growths cause early obstructive symptoms by infiltration but leave the mucous membrane free from actual invasion and therefore do not permit endoscopic biopsy, without which, however, the diagnosis is readily made.

The mucosal cancers are the usual fungating, ulcerating, bleeding, obstructing growths ordinarily encountered.

In all cases, the value of esophagoscopy as a diagnostic procedure is emphasized.

KLAWANS.

Gastroscopy With Flexible Instruments (Gastroscopie avec appareils flexibles).

Baumel, J., Bronchoscopie, Oesophagoscopie et Gastroscopie (April), 1936. Page 121.

The author describes in minute detail the technic of gastroscopy with the flexible gastroscope and describes and illustrates with colored plates the normal and pathologic appearances of the gastric mucosa. This article does not lend itself to abstracting, but should be read by anyone interested in this field.

KLAWANS.

EAR

Histological Research on the Constitutional Fragility of the Lateral Sinus (Ricerche istologische sulla labilita constituzionale del seno laterale).

Fortunato, V. (Roma), Valsalva, 14:686-701 (Dec.), 1935.

Histologic studies of forty lateral sinuses are presented. These postmortem specimens were obtained from individuals of various ages and in whom no auricular disease had existed at any period of their life. The purpose of these investigations, on the mastoid portion of the lateral sinus, was to determine whether the histologic structure of this vein possessed a constitutional weakness of such character as to explain the various pathologic changes observed, in its walls, following an acute or chronic infection or surgical trauma.

The findings are the following:

1. Presence of hyaline degeneration in some zones of the elastic and connective layers of the lateral sinus.

2. Numerous areas of areolar rarefaction in the connective tissue layer, at the mastoid side, forming a reticulum in the fibrous layer.

The author considers the possibility that the hyaline degeneration and the histologic changes in the structure of the wall itself,

found in some cases, may be a locus minoris resistentiae to which any factor, even unrecognized, may be sufficient to cause the breakage of the blood vessel.

SCIARRETTA.

Report Upon 891 Consecutive Cases of Acute Middle Ear Suppuration and Mastoiditis With Intracranial Complications in 139 Cases During the Period 1920-1934.

Fraser, J. S. (Edinburgh) and Halliday, G. C. (Sydney), J. Laryng. and Otol., 51:619 (Oct.), 1936.

Eight hundred and ninety-one cases are reported, sixty of them ending fatally. The authors found that in many cases patients died of purulent meningitis in whom only slight purulent processes existed in middle ear or mastoid; nor was any indication of extradural or other intracranial complication found at operation or on section; nor were the nasal sinuses infected. The possibility is mentioned of a blood infection having a particular affinity for the meninges in the presence of ear suppuration. Only positive blood cultures are of value. On the whole, the authors favor late operations in cases of brain abscess, although there are important exceptions to the rule.

The Vasomotor Control of the Ear.

Fraser, Philip (Birmingham), J. Laryng. and Otol., 51:579 (Sept.), 1936.

This is the report of an investigation of the paths taken by vasoconstrictor impulses to reach the middle and internal ear. Cats were injected with dye by a special technic. Stimulation of the stellate ganglion gave rise to vasoconstriction in the inner, middle and external ear. This was also the case when the superior cervical sympathetic ganglion or the cervical sympathetic trunk were stimulated.

These results were unaffected by decortication of the internal and external carotid arteries.

No vasoconstriction occurs anywhere in the ear on stimulating the stellate ganglion after dividing the cervical sympathetic trunk, thus excluding the possibility of vasoconstrictor impulses reaching the ear from the stellate ganglion via the peri-arterial plexus of the vertebral artery.

An experiment is described which suggests that the vasoconstrictor fibers destined for the area supplied by the first and second divisions of the trigeminal nerve pass through the tympanic plexus of the middle ear.

The bearing of this work on cervical sympathectomy for aural conditions is discussed.

The Indications for the Labyrinth Operation With Special Reference to Acute Diffuse Destructive Labyrinthitis.

Lund, Robert (Copenhagen), J. Laryng. and Otol., 51:425 (July), 1936.

The author describes in detail the various methods of handling diffuse destructive labyrinthitis. During the past twenty-eight years he has greatly modified his own practice. In the first six years he resorted to radical operation; in the next eight he became conservative. During the last fourteen his procedure was based entirely upon spinal fluid examinations. "Normal cerebrospinal fluid . . . contains 0 to 1/3 cells per cm. . . . It will be seen from the above mentioned notes that the contents of the cranium are infected with about the same frequency when labyrinth destruction occurs on account of the cholesteatomatous middle ear suppuration, as in the other diseases of the middle ear, but the infection is of a much milder nature, and the prognosis for the meningeal infection is, therefore, very much better. If the increase of cells is slight (<150/3 cells) you can be sure of almost 100 per cent cure as regards the labyrinthitis (while in the case of acute middle ear suppuration one can be certain of almost 100 per cent mortality.) And even with a turbid cerebrospinal fluid more than half of the patients with cholesteatomatous suppurations are saved by the labyrinthectomy (and we have seen this in only one single case of labyrinth destruction as a consequence of acute middle ear suppuration)."

Treatment of Otosclerosis With Irradiated Yeast (Ein Versuch, die Otosklerose mit bestrahlten Hife zubehandeln).

Charschak, E. (Kiev), Acta Otolar., 23, 3-4:445 (1936).

In this preliminary report much improvement in hearing, lessened tinnitus, and improved calcium were noted in 12 of 15 cases, and it is suggested as an addition to the diet of pregnant otosclerotics.

FENTON.

Tuberculosis of the Middle Ear.

Blegvad, N. Rh. (Copenhagen), Acta Otolar., 24, 1:187 (1936).

While diagnosis of chronic tuberculous otitis media may be very difficult, Blegvad, after twenty years' observation of many patients in Danish sanatoria, maintains that early tuberculosis may always be recognized by dilated and tortuous vessels running from the manubrium to the periphery, without diffuse redness, with the drum membrane otherwise natural in color, often for many weeks or months. Later, diffuse swelling and bulging below, with a characteristic yellowish swelling, complete the picture. Next comes a period of desquamation, followed by two or three perforations which

may coalesce with complete loss of the membrane. In the early stages, before exudation occurs, there may be no complaint beyond slight fullness. There is rarely any associated nasopharyngeal tuberculosis, hence these ear conditions are probably hematogenous.

Treatment is generalized light therapy; of 119 cases (153 ears) two required radical and three simple mastoids, and in two others the ossicles were sequestrated. In one infant the squamous portion of the temporal bone came away as a sequestrum. Paracentesis and inflation should be avoided because of the danger of superadded purulent infection.

FENTON.

Roentgen Therapy of Chronic Purulent Otitis (La roentgenotherapie des otites chroniques purulentes).

Rabinovitch, Z. G. (Rostov), Rev. de Lar., Ot., Rhin., 57:660 (June), 1936.

Two hundred and eighty cases of chronic purulent otitis of from 2 to 20 years' duration, in patients mostly from 6 to 30 years of age, received from one to three series of four treatments ten days apart; the series were repeated every six weeks as necessary. The temporal bone was irradiated at 25 cm. distance over a 6 x 8 cm. field, with 25 per cent filtration HED in children, 30 per cent HED in adults, 120 KV and 3 milliamperes. Eighteen and six-tenths per cent were entirely cured, 20 per cent markedly benefited, and 61 per cent remained unchanged. The improvement is ascribed to stimulation of the formation of connective tissue. The procedure is recommended not only for cases in which operation is contraindicated, but also for speeding up in the cure of fistulæ and delayed postoperative wounds.

FENTON.

The Importance of Otomicroscopy in the Diagnosis and Treatment of the So-called Secretory Middle Ear Catarrh.

Lüscher, E. (Bern), J. Laryng. and Otol., 51:454 (July), 1936.

The author stresses the importance of classifying the various types of discharge in the tympanum. (Six instructive illustrations in color.)

MISCELLANEOUS

A New Classification of the Bones Forming the Skull.

Eagleton, Wells P. (Newark), Arch. Otolaryng., 24:158 (Aug.), 1936.

It is the continuation through life of augmented nutritional properties of growth, largely because of their unique blood supplies from bilaterally placed and freely anastomosing vessels, that gives

rise to the specialized marrow-containing bones of the base of the skull—the base of the sphenoid and, to a lesser degree, the petrous apexes—and to their (a) distinctive reparative activity as well as (b) their specific biochemical resistance against infection. These structures have (1) comparative freedom from infection and (2) specific powers of bone repair; they give a distinctive character to lesions when the structures are attacked by suppuration.

Surgical treatment can bring about cure in a considerable proportion of patients with meningitis, but it must be properly directed, for operation on an inflamed vessel in the bone, which vessel passes from the bone into the dura, does either good or harm. There is no such thing as "harmless exploration" in suppurative disease of the base of the skull. Thus the problem of meningitis is a problem of physiology as modified by infection.

TOBEY.

Palatograms in Deafmutes (Il palatogramma nei sordomuti).

Ciurlo, L. (Milano), Arch. Ital. di Otol., Rino. and Laring., 47:261-288 (April), 1935.

The author reviews the work of previous writers, discusses their methods of preparing a palatogram, and describes his own technic, which is a modification of Rousselot's procedure.

First he makes an exact replica of the patient's palate in plaster of paris, employing the usual dental technic. On this plaster model he carefully places a moistened quadrilateral piece of very thin Japanese silk paper, compressing it between the negative and positive. In this manner he obtains an accurate impression of all the grooves and folds. Then, in the same fashion, a second and third piece of this paper is gued on top of one another. This completes the palatogram, which is allowed to dry, or to hasten this process it may be placed in a slow oven.

The dried paper palate is then removed from its model. Exuberant portions of paper are cut out precisely along the margins of the dental imprints and are finished by a coat of cellulose varnish. When several of these paper palates are prepared, the tongue of the deafmute is covered with a powder composed of cocoa, sugar and gum-arabic, with the aid of a blower. Then the paper palate is fixed in the mouth of the patient, who is instructed to enunciate the words desired for record. The tongue, covered by this colored substance, will leave its imprint on the paper palate, and the palatogram is thus completed and ready for study.

These palatograms are a great aid to deafmute patients, either as an inquiry to the correctness of speech articulation or as a pedagogic method to show the patient the errors which are committed during speech articulation, also to teach him, by comparison with the normal impression, the correct position of his tongue during a normal enunciation. Further, the palatograms taken at regular intervals will record the progress made by the patient while going to a phonetic school.

This article also contains a large number of pathologic and normal palatograms.

SCIARRETTA.

The Illness of the Emperor Frederick.

Colledge, Lionel (London), Brit. Med. J., page 916, November 9, 1935.

Mr. Colledge, in his presidential address to the Section of Laryngology of the Royal Society of Medicine, made it clear that he was neither criticising nor defending those who had charge of the royal patient; his purpose was to study the illness from the pathologic standpoint in the light of modern knowledge.

Sir Morell MacKenzie examined the Crown Prince on May 20, 1887, and found with the mirror a growth about the size of a split pea on the posterior part of the left vocal cord, disappearing from view on phonation. He stated that this showed that it was partly attached to the under surface as well as to the side of the cord—in other words, was partly subglottic. A portion of the tumor was removed on three different occasions, but each time it was reported to show no evidence of malignant changes. In November cancer was diagnosed and the disease ended fatally in June, 1888. Necropsy revealed destruction of the larynx by growth and perichondritis.

The speaker pointed out that the failure to make an earlier diagnosis of cancer was because it was subglottic. The majority of intralaryngeal cancers originate on the middle or anterior portion of one or other vocal cord, but a much less common variety originates beneath the glottis. Isambert described the condition in 1876, but it did not attract much attention until redescribed in recent years by Sir St. Clair Thomson, who drew attention to its grave prognosis. In this case it was evident that while attention had been fixed mainly on the cords themselves and the region above, the tumor was spreading widely and rapidly below the cords. The attempt to remove pieces with forceps by the indirect method when the tumor was subglottic merely resulted in misleading pathologic reports.

GOLDSMITH,
MCLEOD.

The Hemolytic Streptococcus—With Special Reference to Scarlet Fever.

Brewer, Dunstan, Brit. Med. J., page 620, September 6, 1936.

Fatal streptococcal infection has shown an increase during the past fifty years. It is not only as a lethal agent that the streptococcus is important to us; it is the commonest of all causes of temporary inconvenience, and also of permanent defection from normal health. Diseases caused by this organism, including scarlet fever, erysipelas and puerperal fever as well as a miscellaneous group embracing almost every form of sickness from which we suffer are discussed.

The reservoir of the hemolytic streptococci pathogenic to man is the human pharynx: elsewhere it is found only in lesions caused by it. The pathogenic varieties are found in from 1 to 100 per cent of healthy pharynges according to season, period and locality. It is questionable whether they are more frequent in unhealthy or convalescent throats than in healthy throats. They can, of course, always be found in the throats of scarlet fever and streptococcal tonsillitis patients, and for a short time after the reaction has subsided, but it is doubtful if true carriers are more common in those who have reacted than in those who have not. The virulence of the pathogenic streptococci appears to be exalted by passage through a host for other hosts similarly circumstanced. Recently the bacteriologists have reduced the number or varieties of the streptococcus important to human pathology to about a dozen. The feasibility of conferring artificial immunity to this group is therefore promising. But, unfortunately, immunity to streptococcal disease is of a very different order from that to toxemias, to virus diseases and to those dependent upon bacillary invasion. It seems that immunity to the streptococcus is largely local, and strains innocuous at a site where the host has previously reacted to them may be dangerous at a fresh site. Immunization against scarlet fever has been practiced for many years with some success, but the immunity conferred appears to be limited to the toxemia of scarlet fever, and lacks proof of influence upon the liability to other streptococcal diseases. Passive immunity to toxemia can be obtained by anti-scarlet serum, but evidence that this passive immunity protects against streptococcal invasion and septicemia, which are the real dangers, is not encouraging. The writer believes it is unlikely that we shall ever prevent pathogenic streptococcus from living parasitically upon us, although we may look with some confidence for a means of rendering this biologic union harmless. The key to it, he thinks, will probably be found in the study of scarlet fever.

GOLDSMITH.
MCLEOD.

Cavernous Sinus Thrombosis.

Cavenagh, J. B., Brit. Med. J., p. 1195, June 13, 1936.

This paper reviews the cases of cavernous sinus thrombosis from the literature to date and reports one case from the writer's experience. The fact that the veins in this region possess few valves permits not only the rapid invasion of the sinus, which may occur through afferent channels, such as the ophthalmic veins, but also a slower retrograde thrombosis and spread of infection along afferent tracts, such as the internal pterygoid plexus and petrosal sinuses. Herein lies, to a great extent, the explanation of the two main clinical types of thrombophlebitis of the cavernous sinus. First, the acute fulminating invasion, usually associated with septic lesions of the face, and the second type—the chronic compensatory—where the invasion is more gradual and insidious.

The treatment is discussed according as to whether it comprises: 1. major surgery, directed at the cavernous sinus itself; 2. accessory surgical measures dealing with the primary source of infection and the draining of formed abscesses; 3. serotherapy, blood transfusions, etc., in treating the general blood stream infection; or 4. spontaneous recovery in the absence of any form of treatment.

A review of the cases of recovery from cavernous sinus thrombosis shows no more than seven which might be described as the acute infective type. Of this number, three were treated by direct operation on the sinus itself and four by general blood stream therapy. A combination of these two methods, together with ligature of the internal or common carotid artery, is indicated if better results are to be obtained in the future. Approximately thirty cases have been reported of the chronic compensatory type, and of these two have been treated by operation on the sinus itself, one by blood stream therapy, and the majority of the remainder, including a case of the author's, by expectant treatment and by accessory surgical measures for the eradication and drainage of the primary focus, channels of invasion and formed abscesses. A very few cases have recovered spontaneously.

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